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FURTHER OBSERVATIONS ON TUMOR OF THE PINEAL BODY

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During the past twenty years I have had occasion to report either alone or in conjunction with some co-author various observations concerned with the possible physiologic function of the pineal body, together with the pathologic and clinical manifestations of pineal tumors. Concerning the former of these observations, namely, the possible function of this organ, little has been added of recent years. On the other hand, pathologic and clinical observations having to do with tumors of the pineal body have added considerably to the exact knowledge as to the structure of these tumors and as to the clinical syndrome which a patient may present both before and after the age of puberty. The symptom complex in the adult concerns itself wholly with the existence of increased intracranial pressure, together with evidence of localized pressure on the upper portion of the midbrain and on the corpora quadrigemina, which lie directly below the pineal body. Before the age of puberty some patients with a pineal tumor display merely the same evidences of general and localized pressure as those found in adults. On the other hand, in other young persons there is associated with such a tumor an extraordinary degree of premature physical and sexual development, which has been termed macrogenitosomia praecox or simply pubertas praecox. With one possible exception, this syndrome, when it has occurred, has been confined to male children. The reason for its occurrence in some children who have a tumor of the pineal body while it is absent in others is entirely unknown. It is also well recognized that the same precociousness of development may occur in children who have a tumor of the testicles or adrenal glands, and, rarely, it may occur with some condition other than tumor.

No better statement regarding the knowledge of the physiology, pathology and clinical symptomatology of the pineal body can be made

From the Neurosurgical Service of the Lahey Clinic and the New England Deaconess Hospital.

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than that which Dr. Bailey and I quoted in 1928.[1] This quotation was taken from an article by Berblinger [2] published in 1925, in which he said: "In the realm of the pathologic anatomy of the pineal body we must collect experiences and bring together observation after observation. With the increasing number of similar necropsy records capable of being interpreted in the same manner without doing violence to the findings our insight into the essential relationships will deepen." Although the observations which are brought together in the present paper are not concerned with necropsy findings, they are concerned with certain aspects of tumor of the pineal body. In one case the tumor was partially removed and verified at operation, and in the other, although the tumor was not verified either at operation or at necropsy, the growth was almost beyond question a tumor of the pineal body in a young adult showing the striking syndrome of pubertas praecox. In this instance the growth was demonstrated only by its shadow in a ventriculogram.

The first case is reported particularly as an example of what may be accomplished under favorable circumstances either by operation or possibly by a combination of surgical intervention and high voltage roentgen therapy for certain types of growths of the pineal body. That a tumor in this locality is capable of surgical extirpation compatible with survival and a future useful life has been shown particularly by the reports of Dandy [3] in 1921, Van Wagenen [4] in 1931 and Harris and Cairns [5] in 1932. Such experiences, however, are of such infrequent occurrence as to warrant further observations along these lines.

REPORT OF CASES

CASE 1.—*Tumor of the pineal body in an adult. Partial extirpation at operation, after which roentgen therapy was instituted. Recovery, with complete return of vision from near-blindness and normal mental activity, with no evidence of intracranial disturbance one and one-half years subsequently.*

History.—Mrs. E. P., a woman aged 42, was referred to the neurosurgical service by Dr. McKeen of Bookline, Mass., on Oct. 3, 1933, complaining of failing vision and headache. Her family and past history were irrelevant. For a year previous to admission to the hospital she had been having headache, which had become increasingly frequent and severe. The headache had been in both the temporal and the occipital regions and was associated with vertigo. The latter

1. Horrax, G., and Bailey, P.: Pineal Pathology, Arch. Neurol. & Psychiat. **19**:394 (March) 1928.

2. Berblinger, W.: Zur Kenntnis der Zirbelgeschwülste, Ztschr. f. d. ges. Neurol. u. Psychiat. **95**:741, 1925.

3. Dandy, W. E.: An Operation for the Removal of Pineal Tumors, Surg., Gynec. & Obst. **33**:113, 1921.

4. Van Wagenen, W. P.: A Surgical Approach for the Removal of Certain Pineal Tumors, Surg., Gynec. & Obst. **53**:216, 1931.

5. Harris, W., and Cairns, H. W. B.: Diagnosis and Treatment of Pineal Tumors, Lancet **1**:3, 1932.

was aggravated by any change of position. There had been no tinnitus or deafness. The patient had noted that she had become much slower mentally during the previous year, and she stated that her reaction time was much slower than it was a year before. Her memory had been affected, as she often repeated herself, whereas previously she was not accustomed to do this. She also complained of peculiar sensations in the head and of a general "let-down feeling." During the two weeks previous to admission to the hospital her eyesight had failed definitely, although with glasses she was able to read ordinary print.

Neurologic Examination.—The patient was a rather small, frail woman whose general mental reaction seemed fairly normal, and although her answers to questions were prompt and intelligent, she seemed somewhat vague and "in a daze." The pupils were equal but greatly dilated and reacted fairly well to light and in accommodation. It is to be noted that on the first examination the ocular movements were normal and, particularly, that there was no restriction of movement of the eyeballs upward. There was no nystagmus. Examination of the fundi oculi revealed that the outlines of both disks were entirely obliterated, the right disk being elevated 5 diopters and the left, 3 diopters. There was formation of much new tissue over both disks, together with the usual venous engorgement and tortuosity. There was no further abnormality of the cranial nerves, nor were there evidences of a focal lesion in either cerebral hemisphere. There was an intention tremor in the finger-to-nose test on the right. The patient was somewhat unsteady in the Romberg position but did not actually fall. There was likewise some uncertainty in gait, with a tendency to stagger to the right.

The patient was admitted to the New England Deaconess Hospital on October 12, nine days after she had first been examined at the clinic. At this time diplopia was present, but otherwise examination gave results essentially as recorded previously. On October 14 it was noted that the left pupil was slightly larger than the right, and although both reacted to light and in accommodation, the right did not react as well as the left. There were some tremor of the outstretched hands, and a few nystagmoid jerks of the eyes both to the right and to the left. There was also slight tremor in the finger-to-nose test on both the right and the left side. The appearance of the fundi had not changed essentially. Corrected visual acuity was 20/30 minus on the right and the same on the left. The fields of vision were of normal extent, but there was some enlargement of the blind spots.

First Operation.—On October 19 a ventriculogram was carried out. This showed hugely dilated ventricles, from which 130 cc. of fluid was removed and replaced by 110 cc. of air. Subsequent roentgenograms showed the dilated lateral ventricles, with what was taken to be dilatation of the third ventricle. For this reason the ventriculogram was followed by suboccipital craniotomy, on the basis that there was a block, presumably from a tumor in the posterior fossa. This exploration gave negative results except for the demonstration of a large cistern in the posterior fossa, from which fluid in large amounts was evacuated. A median incision through the vermis of the cerebellum did not disclose a tumor.

The patient made a fairly good convalescence from this procedure, and the choked disks receded to about 2 diopters on each side. On the other hand, there developed more or less continual nausea, with vomiting, and on November 7 it was noted that conjugate movements of the eyeballs upward were limited, that convergence in the left eye was poor and that the pupils failed to react to light, although they did react in accommodation. Also some spasticity began to develop in the lower extremities. In view of these new findings, particularly because of the limitation of movement of the eyeballs in an upward direction, the previously

made ventriculograms were reviewed, and at this time a definite shadow was seen bulging into the posterior portion of the third ventricle, this shadow having been overlooked on the original examination (fig. 1). From this study of the plates it was almost certain that the patient had a tumor of the pineal region.

Second Operation.—On November 14 a right occipital craniotomy was performed, and after the dura was opened a transcortical incision was made diagonally through the posterior part of the field, slanting downward and forward toward the posttemporal region. The greatly dilated right ventricle was opened, and the fluid contents were evacuated. The medial ventricular wall was then scrutinized carefully for any possible area of bulging which might represent an underlying tumor. At the posterior end of the body of the ventricle there seemed to be some definite bulging; so at this point the medial wall of the ventricle was

Fig. 1 (case 1).—Ventriculogram showing the dilated lateral ventricles and shadow of the tumor (arrows) bulging into the posterior part of the dilated third ventricle.

incised. After this incision was carried downward for perhaps 1 cm. there was encountered some soft, grayish-red tissue in the form of a rounded mass, which seemed obviously to be tumor. The growth was dissected free from the surrounding brain by gentle use of cotton pledgets, but only its upper pole could be freed in this way (fig. 2). Several pieces of the growth were secured for histologic study and more tissue was removed by suction. The dura was then replaced, leaving a decompression in the temporal region, and the wound was closed.

Microscopic Diagnosis.—The tissue removed was pinealoma (fig. 3).

Course.—The patient had an extremely stormy convalescence from this procedure, the right ventricle being tapped twice daily during the succeeding four

weeks. There was an uneven temperature, which at times was as high as 104 F. It seemed that she was losing ground rapidly and that it would be only a short time before death. The decompression area in the right temporal region became extremely tense but was immediately reduced by ventricular taps and later by lumbar puncture. Cultures of ventricular and lumbar fluid were made repeatedly, but showed no organisms except on one occasion, probably an instance of contamination, when Staphylococcus aureus was present.

On November 22 high voltage roentgen therapy was started, five treatments being given on successive days.

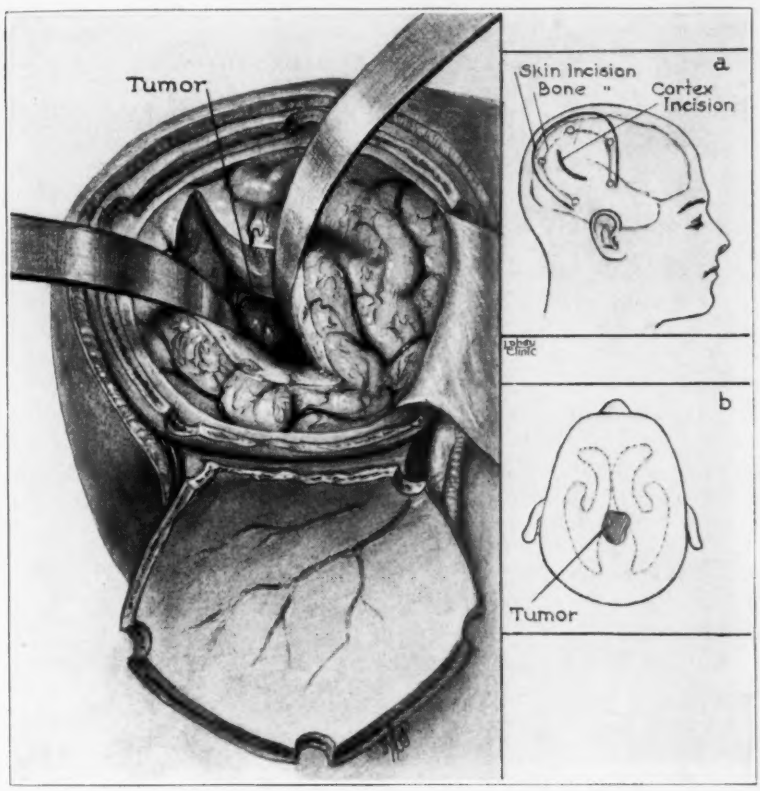

Fig. 2 (case 1).—Drawing of the operative exposure. The cortex has been transected down to the dilated lateral ventricle, and the medial wall of the latter has been incised, disclosing the tumor. Insert *a* indicates the situation of the bone flap and *b*, the approximate relation of the tumor to the ventricles.

The patient continued to lose ground until December 30, when, for the first time, she seemed to start to improve. From about this date the temperature became normal, and all evidences of the previous increase in intracranial tension subsided. By Jan. 3, 1934, when the patient became conscious and rational, it was noted that vision had failed considerably, in that she was able only to count fingers at reading distance. She continued to gain in general physical condition,

was allowed to get up gradually and was discharged on Jan. 18 (fig. 4). By this time she could read the headlines of newspapers, and the choked disks had become nearly flat.

The patient returned to the hospital in May 1934 in order to have contractures in the legs straightened. These had developed during the course of the prolonged serious state when she had not been expected to live. During this time she was given another course of roentgen treatment.

Later Examination (April 10, 1935).—During the previous year the patient had been seen and examined from time to time. She had made steady progress in every way, and except for the partial contractures of the legs would have been normal. Vision had returned to 20/20 in each eye; the pupils reacted to light and in accommodation, and the conjugate movements of the eyeballs upward were restricted

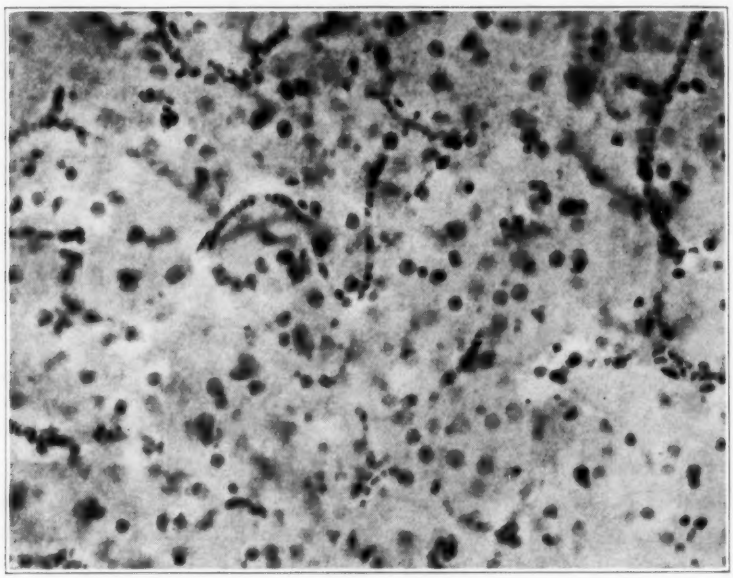

Fig. 3 (case 1).—Photomicrograph of a section of the tumor.

only to the slightest degree. The mental faculties were unimpaired, and there was no bulging of either the suboccipital or the temporal decompression area (fig. 5).

Comment.—There are many points of interest in the history of this patient, but it will suffice to call attention to a few of the more important ones. Primarily, the case is reported as another instance of survival with useful activity following surgical intervention on a tumor of the pineal body. The surgical procedure was the same as that described by Van Wagenen in 1931. In all probability the growth was incompletely removed, but with the institution of roentgen therapy the patient has returned to complete normality with the exception of partial flexion contractures of the legs. There was no evidence of increased intracranial pressure one and one-half years after operation. It is especially to be

noted that two of the striking neurologic signs of tumor of the pineal body, namely, loss of pupillary reaction to light and restriction of ocular movements above the horizontal, entirely disappeared. Another gratifying feature is that although at one time during the illness vision had declined to the counting of fingers, it returned to normal limits.

Fig. 4 (case 1).—A lateral view of the patient before she left the hospital, showing the operative scar.

Fig. 5 (case 1).—Appearance of the patient one and one-half years after the operation.

The second case to be reported cannot by the usual pathologic criteria be regarded as one of verified tumor of the pineal body. Nevertheless, it is reported as an almost certain example of such a growth,

not only because of the clinical syndrome but because of ventriculographic evidence of the tumor itself.

CASE 2.—*Typical picture of pubertas praecox and symptoms of intracranial pressure in a boy; shadow of a presumed tumor in the region of the pineal body seen in the ventriculogram. Decompression followed by roentgen treatment, with recession of intracranial pressure and of some features of precocity.*

History.—C. C., a boy aged 10 years, was referred to the neurosurgical service on May 2, 1934, by Dr. G. W. Ewing of Peabody, Mass. There were no special features about the family or past history. The patient had one sister, aged 13, whose development was normal for her age. During the year previous to admission to the hospital the patient had gained 21 pounds (9.5 Kg.) and his height had become considerably greater, although the exact gain was not known. The father stated that in addition to the increase in height and weight the boy had become extraordinarily well developed and muscular, the voice had changed to that of an adult and the amount of facial and bodily hair had increased greatly. The external genitalia had likewise reached adult proportions. The patient had been in the third grade at school.

For three weeks before admission to the hospital the patient complained of headache, especially in the front and in the back of the head, and for ten days he had double vision. For this reason he consulted Dr. Ewing, who found that there was choked disk bilaterally.

Physical and Neurologic Examination.—The child presented the appearance of a well developed boy aged at least 16, when in fact he was only three months past his tenth birthday (fig. 6). General muscular development was extremely pronounced, and there was abundant bodily hair, which was especially developed on the lower part of the legs and the lower half of the thighs. There was a considerable growth of adolescent hair on the upper lip and cheeks; axillary hair was present, and there was pubic hair of adult type and amount, although of feminine distribution. The external genitalia were of adult appearance and proportions, the flaccid penis measuring 8.5 cm. in length and the testes being about the size of small pecan nuts. The testes were fully descended in the scrotum. The child's voice corresponded to the deep bass voice of an adult.

The pupils were equal and reacted fairly well to light and in accommodation, the right pupil being distinctly less active than the left. There was fairly well marked weakness of the right abducens nerve, making external rotation poor with the right eye. Otherwise, the ocular movements were normal, including conjugate movement upward. A few nystagmoid jerks were present on looking toward either side. The outlines of the optic disks were entirely obliterated, and the disks were elevated between 3 and 4 diopters. There were marked venous engorgement and tortuosity. Visual acuity was 20/50 on the right and 20/20 on the left. The fields were normal.

No other neurologic features were elicited. There was no disparity in the strength on the two sides, and no sensory or reflex changes were noted.

The child's weight was 97 pounds (43.5 Kg.), whereas one year before it had been 76 pounds (34.5 Kg.). The height was 57½ inches (145.9 cm.). The basal metabolic rate was 14. In May 1934, before the patient had had any roentgen treatment, 10 cc. of his urine was injected into a capon. This produced in the capon a free growth of comb, spurs and tail plumes. The same amount of urine was injected into a capon after roentgen treatment, and no changes occurred. The blood pressure was 114 systolic and 70 diastolic, which is possibly a little

high for a boy of the patient's age. The preoperative diagnosis was probable pinealoma.

Operations.—On May 12 a ventriculogram was carried out to confirm the preoperative diagnosis. The plates showed considerably dilated ventricles, with a large, distinct shadow bulging into the posterior portion of the dilated third ventricle (fig. 7).

The child's parents were told that there was a good chance of temporary improvement, perhaps for several years, by the simple procedure of decompression followed by roentgen treatment, whereas a radical attack on the tumor,

Fig. 6 (case 2).—Photograph of the patient before the operation, showing the marked physical overdevelopment.

while more hazardous, offered a better chance of permanent cure. They chose the former procedure.

A bone flap was turned down over the right occipital area, and a decompressive opening was made in the dura in the anterior part of the field over the temporal region. This flap was made, rather than the usual subtemporal decompression, on the basis that at some future time the growth would have to be dealt with by this route. No attempt was made to identify the tumor at this operation.

Course.—Five days postoperatively it was noted that the right pupil did not react to light and the left reacted only slightly. On this day also there was

palsy of the abducens nerve bilaterally, and movements of the eyeballs upward were distinctly limited.

On the twelfth postoperative day the area of decompression had become extremely tense, the boy was drowsy and paresis and hypesthesia had developed on the left side. The pupils were dilated and fixed to light, and upward conjugate movement of the eyeballs was impossible above the horizontal plane.

On May 25, thirteen days after operation, roentgen treatment was started and continued for six days, 900 roentgens being given. After the third treatment, it was noted that there was great improvement in the strength of the left side and that the area of decompression, although still full, was definitely less tense. From this time the patient rapidly became better, sat up in a chair and then

Fig. 7 (case 2).—Ventriculogram, with the arrows pointing to the shadow of the tumor bulging into the posterior end of the dilated third ventricle.

was up and about the ward. He was discharged to his home on May 31, nineteen days after operation.

On June 14 the patient was brought to the clinic for a check up. He had not had headache, and the area of decompression was soft, although somewhat full. There was still about 1.5 diopters of choking of each optic disk. The pupils were equal and reacted to light and in accommodation, and upward conjugate movements were normal. There were left homonymous hemianopia and persistent nystagmus to the right and left.

Later Examination.—On September 24 it was reported that the boy had led a normal life through the summer. He had lost 5½ pounds (2.6 Kg.) since the operation and roentgen treatment, his present weight being 91½ pounds.

It was noted that the patient had lost a great deal of the growth of hair on the face, body and legs, although the pubic hair was unchanged. There were likewise no changes in the voice or in the size of the external genitalia. There was, however, a distinctly more youthful appearance of the face (fig. 8), a fact which had been noted especially by the parents.

The patient was seen at intervals up to April 10, 1935. In December 1934 the headache returned, and the area of decompression began to bulge. A series of roentgen treatments caused rapid recession of these signs. The same condition has just recurred at the time of writing, and another series of roentgen treatment has been completed. The results of this are not yet apparent. The pupillary and oculomotor signs have remained normal, and, except for the two brief periods cited, the boy has led an active and normal life.

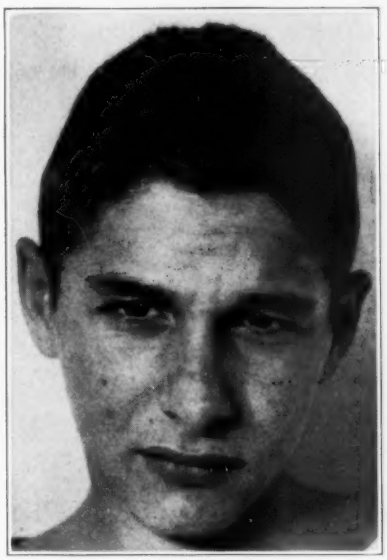

Fig. 8 (case 2).—Appearance of the patient five months after decompression and roentgen treatment. Note the absence of internal strabismus on the right and particularly the more youthful expression of the face.

Comment.—There are three particular points of interest in this case: First, although the growth was not verified pathologically, it was almost certainly another example of a tumor of the pineal body in a male child who exhibited outspoken evidences of physical precocity. Second, it is of special interest that after the boy had received roentgen treatment directed toward the supposed tumor of the pineal body, there was not only a subsidence of the symptoms of intracranial pressure but a definite and really remarkable return to a more naturally childlike appearance. This cannot be brought out satisfactorily in the figures, but it was evident when the child was seen before and after treatment.

It was noted immediately by the parents, even before any comment had been made by the physician. Third, it would appear that roentgen therapy applied to this tumor, although effective for several months after each series of treatments, is much less lasting than in the adult in case 1. In all probability the child will have to submit to radical surgical intervention in the near future.

SUMMARY

Two additional cases of tumor of the pineal body are reported. In one, that of an adult, the growth was partially removed and verified at operation. Subsequent to operation and roentgen therapy the patient had remained well and in active life for one and one-half years, with no return of symptoms of intracranial disturbance.

In the second case, that of a boy 10 years of age, the tumor was verified only by ventriculography. This child presented the typical syndrome of Pellizzi, namely, macrogenitosomia praecox. This patient also had had a period of normal activity for a year following decompression and roentgen therapy. It is of particular interest that after this therapy he became more normally youthful in appearance. This is at least suggestive of some possible function of the pineal body concerned with the regulation of secondary sex characteristics.

DISCUSSION

DR. ERNEST SACHS, St. Louis: Dr. Horrax has, I think, drawn attention to one extremely important point with which I for one was not familiar, namely, that the tumors of the pineal body apparently are highly radiosensitive, which is significant from the therapeutic standpoint. I had the opportunity of reading Dr. Horrax's paper before I came here and therefore reviewed my own cases; I find that since 1921 there have been eleven cases of tumor of the pineal body in this series. Operation was performed in ten, with a successful result in only one case. One patient, who was operated on nine years ago, with a cyst in the region of the pineal body, is well today. Although the other nine patients who were operated on succumbed, one survived two weeks after operation and then died of pneumonia. The eleventh case was that of an infant, aged 2 months, in which the diagnosis was not made until after death. The case was thought to be one of hydrocephalus; in fact, the child did have marked hydrocephalus. High voltage roentgen therapy was used for a prolonged period because it was thought that there was a tumor of the posterior cerebral fossa; in spite of intensive roentgen treatment the tumor continued to grow, and at autopsy, about six months later, there was no evidence of effect from the therapy.

There are two other points I wish to emphasize concerning my own series of cases: First, in the eleven cases there was not one of pubertas praecox. Second, in six of the eleven cases the one outstanding symptom which Dr. Horrax has referred to was present, namely, inability of the patient to look upward, which I think every one interprets as evidence of pressure on the anterior corpora quadrigemina. As regards pubertas praecox (I use that word in preference, because I cannot pronounce the other long name), I know of no instance of a gland or a structure which has a certain function, such as the pineal gland has had, that is, a cyclopean eye in lower forms of animals, which undergoes a change and assumes a different function. I hope that Dr. Horrax will have something

to say about that in his concluding remarks. Therefore, it is difficult for me to understand how the symptom of pubertas praecox can be attributed to the pineal gland or to the cells of the pineal gland. I hope that Dr. Horrax or some one else will correct me if I am mistaken in that statement.

DR. SMITH ELY JELLIFFE, New York: Dr. Horrax gave me the privilege of reading his paper. Tumors of the pineal body are still rare enough to constitute curiosities, and the syndrome is diverse enough to provoke inquiry, discussion and surprise. These tumors have been known a long time. The first systematic collection of reports of such tumors, made by Pearce Bailey and myself in 1911, contained sixty cases, beginning with one that was reported in 1800. Shortly afterward Dr. Horrax entered the field, which he has so well cultivated since. In 1927 Haldeman listed one hundred and thirteen cases, and since then thirty or forty cases have been added.

Fränkl-Hochwart made the initial synthesis, which Marburg has made more precise and to which Horrax and Bailey have made definite contributions. The present status, I think, may be stated briefly somewhat as follows: Little that is new has been added to the embryologic studies of Tilney and Warren and of Krabbe. The Spanish school has added somewhat to the histologic details. The studies of Pines, Greving and others are following up the connections of the diencephalic fiber tract.

I cannot find any tangible or definite unequivocal facts concerning what part the epiphysis plays in the general functions of the body. I cannot go so far as Dandy, who claims that it is of no use; yet I find little definite concurrence of opinion as to any essential activity. Certain animals have only mere vestiges of the pineal body, notably, the alligator, whale and elephant, yet it is a highly vascularized body and goes through definite embryologic alterations (notably studied by Krabbe).

The physiology of the pineal body seems as vague as the idea that it is the supposed home of the soul, a doctrine falsely attributed to Descartes, which Marburg has shown to be a gross error. If it has an endocrine function, my survey of the field leaves me a benevolent skeptic, even in view of the newer claimed and disclaimed estrogenic function, which Dr. Horrax has referred to with reference to the experiments with capons. Commercial extracts of the pineal gland are, I believe, of no value.

It should be remembered that Pellizzi founded his syndrome on clinical material only, without any observations at autopsy. Still, pubertas praecox needs to be explained and also the fact that it occurs practically only in males. It is a definite condition which is still unexplained, even if it is interpreted as being due to pressure on unknown hypothalamic structures, thereby setting gonadal, adrenal or other complexes into activity.

The syndrome attendant on new growths, however, is still enticing, as new signs, often very obscure, are being gathered into the general picture. Rarer signs indicating narcolepsy, diabetes, cerebellar involvement and other associated conditions are being added here which are of interest in conjunction with or isolated from the commoner signs arising from pressure on the corpora quadrigemina and from direct pressure, particularly those seen in Parinaud's syndrome. These neurologic and metabolic signs are making localization more definite and surgical intervention more practical, as the excellent work of Horrax and his associates has again demonstrated.

The cachexia often noted still needs explanation. I am desirous of learning more of possible photoreceptor functions as related to melanin and cutaneous chromophore factors, a field once entered by McCord and apparently neglected. Here again probably the answer may come from the hypothalamic regulation of fat metabolism.

In conclusion, I can only register my congratulations on the recent work here represented, especially as to the highly satisfactory advances that have been made in the surgical field, which, combined with roentgen therapy, offers additional evidence of the fruitful advances made by the neurosurgeon.

DR. WALTER TIMME, New York: Far be it from me to embarrass neurosurgeons or even psychoanalysts by asking them for an explanation of macrogenitosomia praecox. They are so imbued with their own subject that I am afraid that the advances in endocrinologic study have eluded them. It is still more painful to ask such a personal friend as Gilbert Horrax, who reports a case which is indubitably one that has to do with the endocrine factors of a disturbed pineal body, whether he has changed the view which he assumed with Percival Bailey some years ago before this association, that the pineal gland was without any potentialities whatever in the endocrine field.

I must ask Dr. Jelliffe whether he has read the recent monograph on the pineal gland by Calvet, which appeared a year ago, and, if so, whether that made any impression on him.

DR. SMITH ELY JELLIFFE, New York: I have read the article and would refer Dr. Timme to the review in the *Journal of Nervous and Mental Disease* (**81**:713 [June] 1935).

DR. WALTER TIMME, New York: Second, may I also ask whether Dr. Sachs has read the embryologic exposition of the structure of the pineal body or of the epiphysis cerebri by Warren and Tilney? If so, he will see that it is not necessarily the deterioration of a function that produced an endocrine organ, but it is a parapineal structure which happens to be in juxtaposition to the pineal body that shows glandiferous structure capable of producing a secretion. Whether the pineal body itself is involved is not certain. But why an organ should not assume in the course of eons of time, millions of years (I ask Dr. Jelliffe to support me), a completely different function, depending on intercurrent mutations, I do not see. I should like those questions to be answered. An elephant may not have a pineal gland, as Dr. Jelliffe asserts, but then an elephant represents a beautiful case of macrogenitosomia praecox!

DR. GILBERT HORRAX, Boston: I think that Dr. Jelliffe has answered Dr. Sachs sufficiently regarding the histologic evidence for glandular function of the pineal body. I chose to call it the pineal body rather than the pineal gland because I still believe it is not known that it has a definite glandular function.

I am well aware of the work of Tilney and Warren, who demonstrated glandular cells in the pineal body in prenatal life, but I do not believe that any one has demonstrated such cells in postnatal life. Furthermore, I should be glad to believe that the pineal body has an endocrine function, but I cannot quite bring myself to think so. It may be that there are in this parapineal region glandular cells which affect other members of the endocrine chain, because, as all know, the same syndrome of pubertas praecox (I will not attempt the other word for the moment) occurs in cases of tumor of the adrenal gland, tumor of the testis and tumor of the ovary. Furthermore, it occurs in cases of tumor in the region of the third ventricle, not very close to the pineal body. I have here a monograph by Le Marquand in which he reports a case of pubertas praecox in a boy associated with tumor of the floor of the third ventricle at the base of the brain, nowhere near the pineal body. Furthermore, it may occur in cases of tuberous sclerosis. In one instance, I believe, it has occurred in a girl, the only instance in a girl as far as I know, in whom hypoplasia of the pineal body was noted at autopsy at the age of 24. The case had been studied for many years.

I report these cases merely for what they are worth. I agree with Dr. Jelliffe that there is as yet no absolute knowledge about this matter, but it does seem rather striking, as he has said, that the two largest mammals have no pineal body!

AMYOTROPHIC LATERAL SCLEROSIS WITH INVOLVEMENT OF POSTERIOR COLUMN AND SENSORY DISTURBANCES

A CLINICOPATHOLOGIC STUDY

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Amyotrophic lateral sclerosis, correctly regarded as a disease of the motor system, occasionally involves other neural structures, and cases have been recorded in which it was associated with mental symptoms [1] and sensory disturbances of both peripheral [2] and central nature. The question always arose whether the changes in the sensory pathways formed part of the picture of amyotrophic lateral sclerosis or were chance associations in the course of Charcot's disease. Another question is whether or not the degeneration of the pyramidal tract, posterior column and anterior horn cells is the result of a combined system disease, possibly on the basis of pernicious anemia. The following two cases in which degeneration of the posterior columns was present in clinically and pathologically typical syndromes of amyotrophic lateral sclerosis illustrate once more the occurrence of sensory disturbances in the "pure" motor syndrome.

REPORT OF CASES

CASE 1.—M. F., a man aged 51, was admitted to the Montefiore Hospital on Dec. 14, 1929 with a history of numbness and stiffness in the left hand, beginning in November 1928, which gradually spread to the right hand and later involved both lower extremities. In July 1929 he was unable to recognize objects placed in his hand, and speech became hesitant. The past history was irrelevant, and the general medical condition was normal.

Neurologic Examination.—The gait was spastic; there were bilateral ataxia and slight irregular intention tremor of the right upper extremity. All deep reflexes, especially those of the upper extremities, were markedly increased; the abdominal

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From the Neurological Division and Neuropathological Laboratory, Montefiore Hospital.

Read by title at the Sixty-First Annual Meeting of the American Neurological Association, Montreal, Canada, June 3, 1935.

1. Wechsler, I. S., and Davison, C.: Amyotrophic Lateral Sclerosis with Mental Symptoms, Arch. Neurol & Psychiat. **27**:859 (April) 1932.

2. Wechsler, I. S.; Brock, S., and Weil, A.: Amyotrophic Lateral Sclerosis with Objective and Subjective (Neuritic) Sensory Disturbances, Arch. Neurol. & Psychiat. **21**:299 (Feb.) 1929.

reflexes were present; there was a Babinski sign bilaterally. The interosseus muscles, thenar eminences and muscles of the forearms were atrophied. Fibrillations were not noted, but all the muscle groups showed marked myotatic irritability. Voluntary power was reduced in all muscle groups, in the left more than in the right, and more in the upper extremities. Sense of position was markedly impaired in the fingers of both hands but spared in the toes. There was difficulty in recognizing objects in both hands, although the patient could appreciate relative size, shape, weight, form, smoothness, etc. There was also some impairment in two point discrimination in the fingers. Pain, temperature and touch sensations were intact. All the cerebral nerves were normal. Speech was somewhat slow.

Course.—The patient's condition, except for occasional fibrillary twitchings of the tongue without atrophy of its muscles, remained unchanged. He died of bronchopneumonia on April 5, 1931.

Laboratory Data.—The blood, gastric contents, urine and spinal fluid were normal. Serologic tests with the blood and spinal fluid gave negative results.

Autopsy.—Gross Examination: The frontal, motor and postcentral convolutions of the brain were shrunken, and their fissures showed wide separation. On cutting the brain coronally in 1 cm. slabs no gross changes were observed. In the spinal cord the crossed pyramidal pathways appeared translucent.

Microscopic Examination: Frozen and pyroxylin (celloidin)-embedded sections from the various convolutions, diencephalon, midbrain, pons, medulla oblongata and spinal cord were stained by the myelin sheath, cresyl violet, sudan IV, Bielschowsky and Victoria blue methods.

Precentral and Postcentral Convolutions: The giant pyramidal cells of Betz of the precentral convolutions were diminished in number and slightly swollen. They showed lack of Nissl substance and eccentrically placed nuclei. Pyknosis and atrophy of ganglion cells, as seen in chronic cell changes, were also encountered. There was a slight increase in the microglia cells. In sections stained for fat, the ganglion cells showed fatty deposits. The pyramidal cells of the postcentral convolutions, except for an occasional diseased cell, appeared normal.

Midbrain, Pons and Medulla Oblongata: In these sections, except for slight pallor and occasional disintegration of some of the fibers of the pyramidal tract, little change was noticed. In the region of the crossing of the pyramids the pallor and disintegration of fibers were more prominent. Some of the ganglion cells of the nucleus of the twelfth nerve were shrunken and pyknotic and had long corkscrew processes.

Spinal Cord: In myelin sheath preparations there was marked demyelinization of the crossed pyramidal tracts extending from the cervical to the sacral segments (fig. 1). In addition to this, at the first and second dorsal segments there was demyelinization of the posterior columns, the fasciculi cuneati being more involved than the fasciculi graciles (fig. 1). The dorsospinocerebellar tract and the dorsolateral fasciculus (Lissauer) were also involved at this level. In the eighth cervical segment only the fasciculus cuneatus was affected (figs. 1 and 2); this apparently was an ascending degeneration. All the other cervical segments did not show degeneration of the posterior columns. The cord did not have a honeycomb appearance. The myelin sheaths in the demyelinated areas were completely destroyed (fig. 3*A*); the few which remained were swollen and fragmented. In fat preparations marked lipoid accumulation of fat in the destroyed areas was noted. In sections stained by the Bielschowsky method, the axis-cylinders in the demyelinated areas showed marked destructive changes (fig. 3*B*); the few which could be seen were disintegrated and swollen and had a corkscrew appearance. In

sections stained with Victoria blue the destroyed areas were replaced by a dense glia scar (fig. 4). In cresyl violet preparations the anterior horn cells throughout the cord were decreased in number; those which were preserved were shrunken and pyknotic and had long as well as corkscrew processes (fig. 5*A* and *B*). In some of the dorsal segments the anterior horn cells of the ventromesial group showed clumping of Nissl substance and chromatolytic changes. Pigmentary atrophy was also noticed. The process was more extensive in the eighth cervical and first dorsal segments; the nerve cells of the ventrolateral group were more involved than the other groups. The anterior, the posterolateral spinal and intraspinal arteries did not show atherosclerotic changes. There were no evidences of an inflammatory reaction. The peripheral nerves were normal.

Comment.—Except for the impairment in sense of position in the fingers of both hands and in two point discrimination and the astereognosis, the symptomatology was typical of the syndrome of amyotrophic lateral sclerosis. The combination, however, of posterior

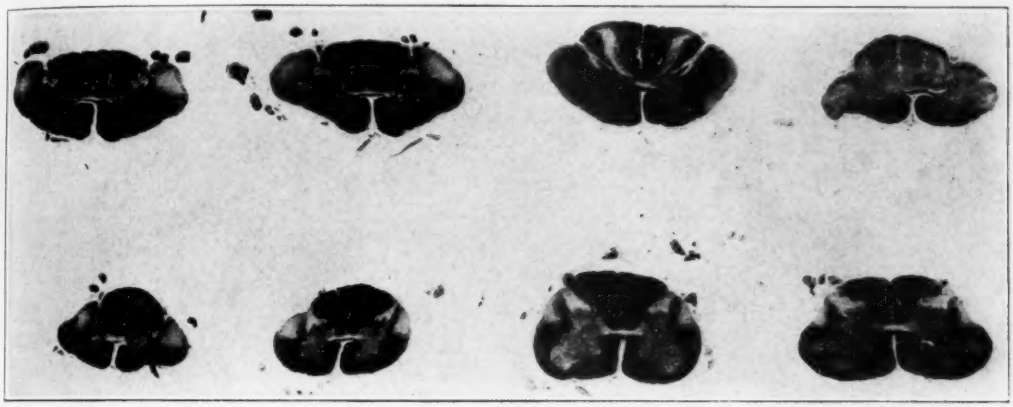

Fig. 1 (case 1).—Demyelinization of the crossed pyramidal tracts extending from the cervical to the sacral dermatomes. Notice the additional demyelinization of the posterior columns, dorsospinocerebellar tracts and dorsolateral fasciculus in the region of the second dorsal segment and the involvement of only the fasciculi cuneati in the eighth servical segment. Myelin sheath stain.

column and pyramidal tract signs raised the question of subacute combined degeneration. Atrophy of muscles, although not usually found in subacute combined degeneration, have been described by Holmes,[3] Stone[4] and others. The normal blood picture and gastric contents obviously ruled out such a possibility and compelled a clinical diagnosis of amyotrophic lateral sclerosis, which was confirmed by histologic

3. Holmes, Gordon: A Case of Combined Degeneration of the Spinal Cord with Amyotrophy, Rev. Neurol. & Psychiat. **11**:76, 1913.

4. Stone, T. T.: Subacute Combined Degeneration of the Spinal Cord with Chronic Changes in the Anterior Horns of the Cervico-Dorsal Region, J. Nerv. & Ment. Dis. **73**:41, 1931.

evidence of typical involvement of the anterior horns. The demyelinating process in the posterior columns of the lowermost cervical and the first two dorsal segments, which accounted for the astereognosis and the loss of the sense of position, is more difficult to explain. The type of destruction of the crossed pyramidal tracts which was present in all the segments of the cord could also be observed in the medulla oblongata and pons. In subacute combined degeneration, involvement of the posterior columns and pyramidal tracts at only one level is rare. Furthermore, involvement of the pyramidal tracts alone throughout the cord, as far as we know, has never been described in lesions of the

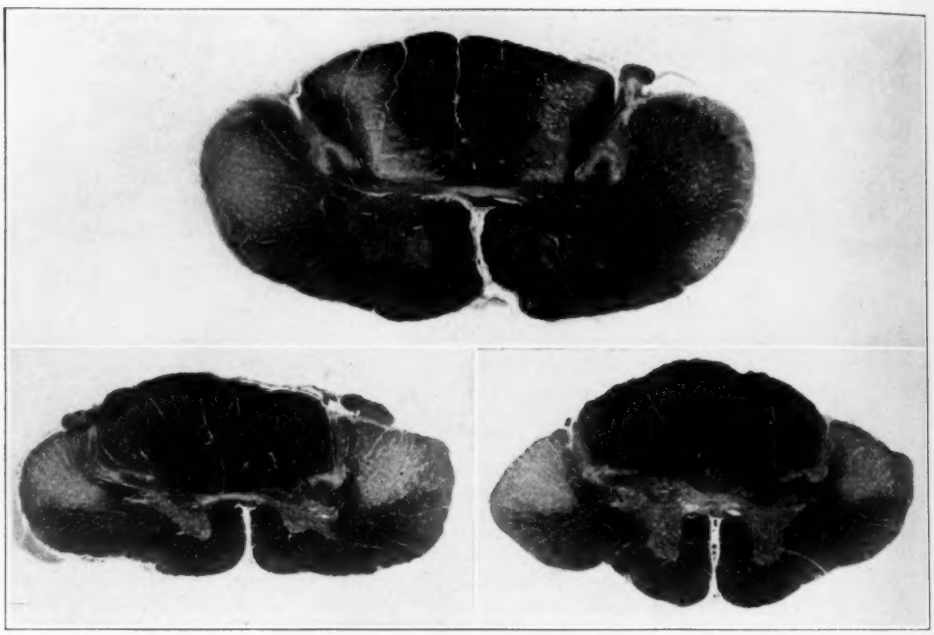

Fig. 2 (case 1).—Demyelinization of the fasciculi cuneati in the eighth cervical segment and of the crossed pyramidal tracts in this and other segments. Myelin sheath stain.

cord due to pernicious anemia. The destroyed myelin sheaths and axis-cylinders were replaced by an intense glial scar (fig. 4), a process not seen in subacute combined degeneration unless the patient received liver therapy (Davison), which was not so in this case, nor was there evidence of inflammatory or arteriosclerotic changes in the vessels.

Case 2.—R. Z., a woman aged 58, was admitted to the Montefiore Hospital on Feb. 7, 1930. In the spring of 1929 she fell and struck her right shoulder and began to have cramplike burning pain in the muscles and joints. Roentgen examination of the shoulder gave negative results. The pains became more severe,

more frequent and of longer duration and spread to all extremities. Later, weakness and atrophy of the muscles of the upper extremities and spasticity of the lower extremities developed. The past history of the patient and the family history were irrelevant, and general medical examination gave negative results.

Neurologic Examination.—The patient was unable to walk. There was weakness of all muscles groups in both the upper and the lower extremities; the triceps muscles were the least affected. The lower extremities were spastic, and there was bilateral footdrop. The muscles of the hands and forearms were atrophied; these, as well as the biceps, deltoids and muscles of the neck and chest were the seat of fibrillations. The deep reflexes of the upper and lower extremities were exaggerated; there were positive Babinski and allied signs. There were hypesthesia and hypalgesia in the distal portions of the extremities and absence of vibratory sensation in the

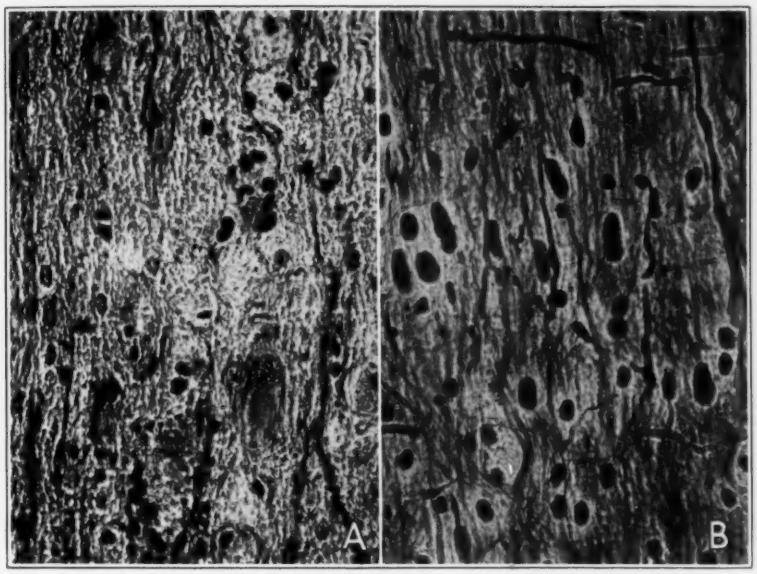

Fig. 3 (case 1).—*A*, fragmentation of the myelin sheaths in the areas of degeneration. Myelin sheath stain; reduced from × 200. *B*, disintegration and swelling of axis-cylinders in the area of degeneration. Bielschowsky stain; reduced from × 200.

lower extremities; sense of position was intact. The tongue was atrophied and showed fibrillations, as did the perioral muscles.

Course.—The condition became progressively worse; pulmonary edema developed, and the patient died on Sept. 13, 1930.

Laboratory Data.—Roentgen examination of the spine showed a moderate degree of spondylitis. Examination of the blood showed: hemoglobin, 78 per cent; red cells, 3,879,000; white cells, 9,400; polymorphonuclear leukocytes, 59 per cent, lymphocytes, 30 per cent, and large monocytes, 1 per cent. There were no nucleated red cells. An analysis of the gastric contents was not made. All other laboratory data were normal.

Autopsy.—Gross Examination: The frontal convolutions of the brain were atrophic, and the frontal and left central fissures were prominent. The blood vessels were normal. The brain was cut coronally. There was a slight generalized internal hydrocephalus. The dura of the spinal cord had a copper color, and the vessels were slightly injected. In transverse sections of the cord the lateral pyramidal tracts appeared translucent.

Microscopic Examination: Frozen and pyroxylin (celloidin) embedded sections from the frontal lobes, motor cortex, pons, medulla oblongata and spinal cord were stained by the same methods as in the preceding case.

Frontal and Precentral Convolutions: Sections from the frontal convolutions showed a decrease in the number of ganglion cells of the third layer; these stained poorly and disclosed loss of Nissl substance and eccentrically placed nuclei. The microglia and protoplasmic astrocytes in the third, fifth and sixth layers were

Fig. 4 (case 1).—Dense glia scar replacing the destroyed myelin sheaths and axis-cylinders. Holzer stain; reduced from × 200.

slightly increased in number. In the motor area the nerve cells of the third layer disclosed the same changes as those in the frontal convolutions. The giant pyramidal cells of Betz were diminished in number, shrunken and pyknotic. The ganglion cells of the sixth layer presented chronic changes.

Pons and Medulla Oblongata: Sections of the pons and medulla oblongata showed demyelinization of the pyramids. On higher magnification the myelin sheaths were found to be disintegrated and slightly swollen. A few necrobiotic foci limited to the pyramids were also found. The nerve cells of the hypoglossal nuclei were fewer than normal and showed shrinkage, pyknosis and corkscrew processes. All other nerve cells in the medulla oblongata were well preserved.

Spinal Cord: In myelin sheath preparations the crossed pyramidal tracts were demyelinated throughout all segments of the cord (fig. 6). At several levels the direct pyramidal tracts were also demyelinated. In addition the sections between

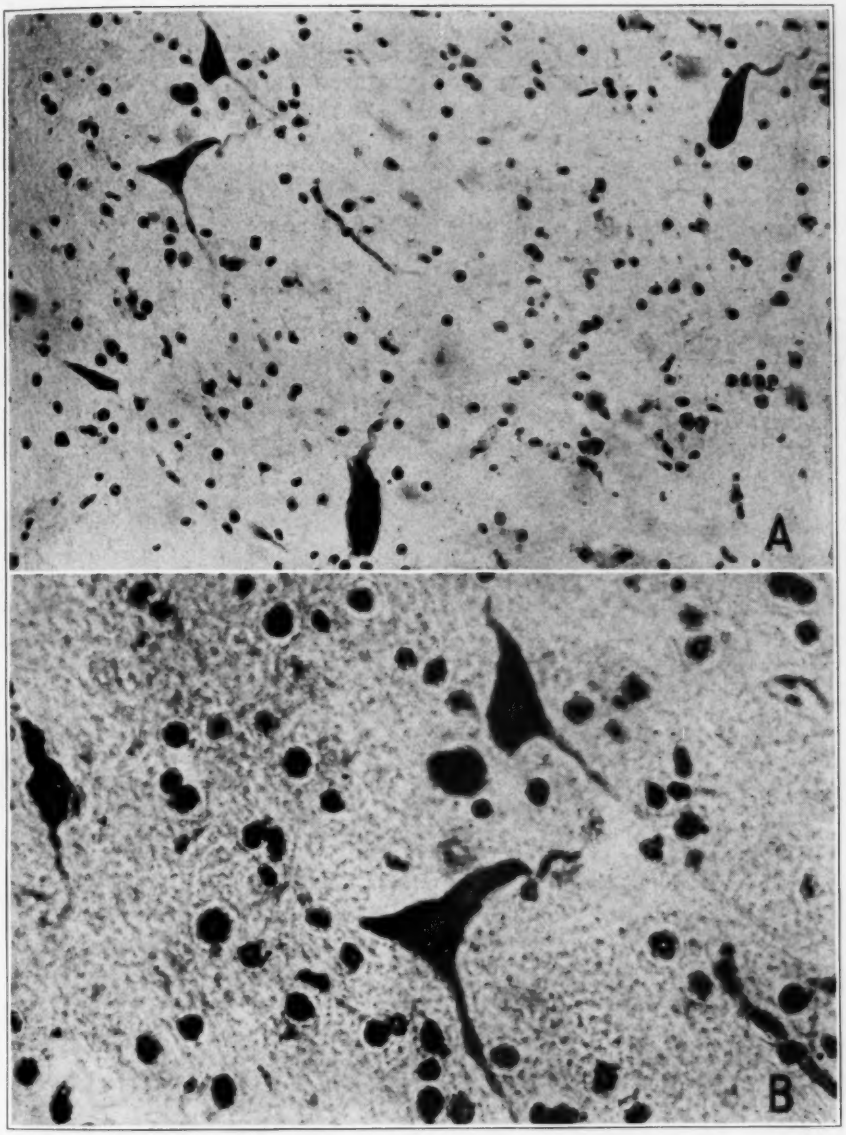

Fig. 5 (case 1).—*A*, diminution in number, shrinkage and pyknosis of anterior horn cells. Cresyl violet; × 240. *B*, same as *A*; × 480.

the tenth and eleventh dorsal segments disclosed demyelinization of the posterior columns, dorsolateral fasciculi and dorsospinocerebellar pathways and part of the spinothalamic tracts (fig. 6). The greatest amount of demyelinization was at the eleventh dorsal segment. The fasciculi graciles were more involved than the fasciculi cuneati. Sections above the tenth dorsal segment disclosed an ascending degeneration of the fasciculi graciles. With higher magnification the myelin sheaths in the demyelinated areas appeared disintegrated and swollen. Marked increase in the number of compound granular corpuscles and fat accumulations were observed with the sudan IV stain. In Bielschowsky preparations the axis-cylinders appeared fragmented and swollen; some had corkscrew processes. In sections stained with Victoria blue there was an increase of glia fibers in the affected areas. The cord did not have a honeycomb appearance. The spinal vessels

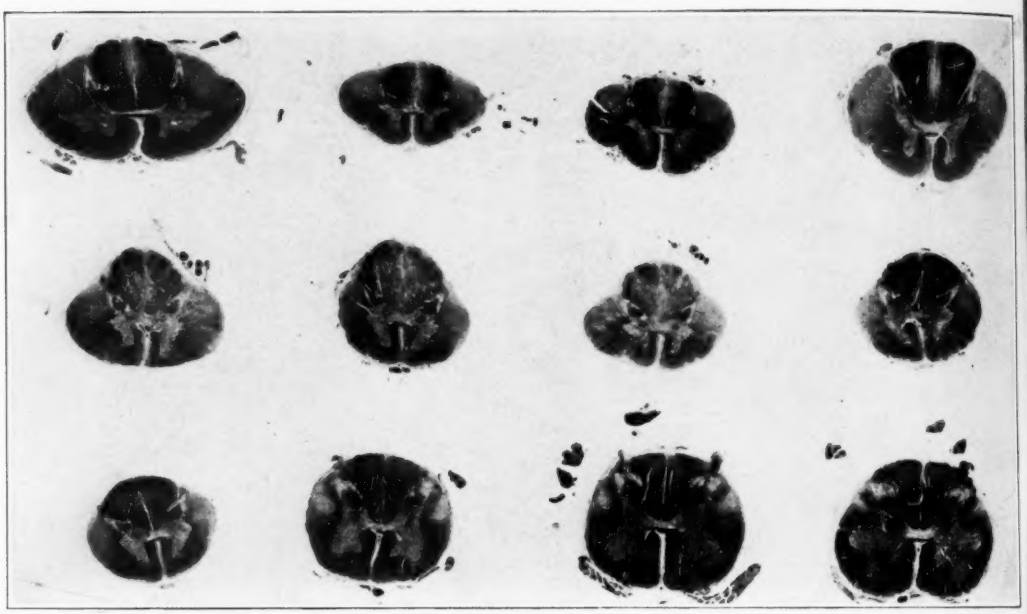

Fig. 6 (case 2).—Demyelinization of the crossed pyramidal tracts from the cervical to the sacral segments. Notice the demyelinization of the posterior columns, dorsolateral fasciculus and dorsospinocerebellar and spinothalamic pathways in the region of the eleventh dorsal segment. The sections above the tenth dorsal segment disclose an ascending degeneration of the fasciculi graciles. Myelin sheath stain.

were normal. The anterior horn cells in cresyl violet preparations were decreased in number, shrunken and pyknotic, and contained darkly stained nuclei; pigmentary atrophy was also noted. The ventrolateral group seemed to be most involved. The nerve cells of the lateral horns were swollen and devoid of Nissl substance, and their nuclei were displaced to the periphery. The peripheral nerves were normal.

Comment.—Clinically, this patient presented a clearcut picture of amyotrophic lateral sclerosis with some questionable radicular pains,

absence of vibratory sensation in the lower extremities and hypalgesia and hypesthesia in the distal portions of the extremities. The histopathologic changes were those seen in amyotrophic lateral sclerosis. However, the demyelinization of the lateral pyramidal tracts and of the posterior columns, spinocerebellar and part of the spinothalamic pathways between the tenth and eleventh dorsal segments gave the cord an appearance such as is observed in subacute combined degeneration. It was merely an appearance, for in the latter disease one rarely observes involvement of the posterior columns and pyramidal tracts at one or two levels only. The absence of the honeycomb appearance in the cord and the increase in the glia are further negative proof. It is to be emphasized that the pyramidal tracts were demyelinated throughout all segments of the spinal cord, medulla oblongata and pons. It may be concluded that whatever factor was responsible for the amyotrophy was also the cause of the degeneration of the other pathways. It is difficult to explain why the destruction should have occurred at only one level of the cord.

GENERAL COMMENT

The two cases presenting unusual histopathologic features, namely, involvement of the posterior columns and other pathways at one or two levels, were recognized clinically as instances of amyotrophic lateral sclerosis. In the first case, the possibility that the condition was subacute combined degeneration was considered because of the astereognosis and vague sensory disturbances noted in addition to the usual picture of amyotrophic lateral sclerosis. Negative results from gastric analysis, a reliable diagnostic criterion against pernicious anemia, definitely ruled out the latter. In previous cases described by Anglada,[5] Wechsler, Brock and Weil [2] and Hassin,[6] radicular pains raised the questions of extramedullary tumor and neuritis. In some of the cases described in the literature the patients were subjected to laminectomy. Hassin's patient did not show signs of involvement of the posterior columns before the laminectomy, but at necropsy the cord showed degeneration of the columns of Goll in the cervical, dorsal and lumbar regions. This was in contrast to the observations in our cases. Hassin also pointed out the absence of the spongelike appearance of the degenerated areas, a process so often seen in subacute combined degeneration. It should be pointed out that the peripheral nerves and posterior roots were not affected in Hassin's case or in ours. This definitely rules out the question of an ascending degeneration. In our first case the ascending

5. Anglada, J.: Sclérose latérale amyotrophique ascendante avec manifestations douloureuses et paraplégie en fléxion intense, Montpellier méd. **32**:73, 1911.

6. Hassin, G. B.: Amyotrophic Lateral Sclerosis Complicated by Subacute Combined Degeneration of the Cord, Arch. Neurol. & Psychiat. **29**:125 (Jan.) 1933.

degeneration of the fasciculus cuneatus limited to only one level emphasizes this point. In the second case the demyelinization of the fasciculus gracilis extended farther up.

The rarity with which one meets involvement of sensory tracts in amyotrophic lateral sclerosis is attested by the fact that Oppenheim [7] only once observed encroachment on the posterior horns. Posterior column changes in amyotrophic lateral sclerosis were, however, noted by Moeli,[8] Charcot and Marie,[9] Hektoen,[10] Pilcz,[11] Dercum and Spiller,[12] Czylark and Marburg,[13] Mott and Tredgold,[14] Lejonne and Lhermitte,[15] Holmes,[3] Babonneix and Widiez,[16] Hassin,[6] and Dagnélie and Cambier.[17] The case described by Babonneix and Widiez most likely was not one of amyotrophic lateral sclerosis, but was due to syphilis.

While the association of sensory disturbances with a syndrome of amyotrophic lateral sclerosis raises justifiable doubts, the diagnosis is not difficult. In such cases the suspicion of subacute combined degeneration can be eliminated by the Castle test [18] and gastric analysis. If free hydrochloric acid is present it is safe to rule out pernicious anemia. A negative blood picture alone does not rule out the diagnosis since the

7. Oppenheim, H.: Text-Book of Nervous Diseases, translated by A. Bruce, Edinburgh, O. Schultze & Co., 1911, vol. 1, p. 228.

8. Moeli, C.: Ein Fall von amyotrophischer Lateralsklerose, Arch. f. Psychiat. **16**:718, 1879.

9. Charcot, J. M., and Marie, P.: Deux nouveux cas de sclérose laterale amyotrophique suivis d'autopsie, Arch. de neurol. **10**:1, 1885.

10. Hektoen, L.: Amyotrophic Lateral Sclerosis with Bulbar Paralysis and Degeneration in Goll's Columns, J. Nerv. & Ment. Dis. **20**:145, 1895.

11. Pilcz, A.: Ueber einen Fall von amyotrophischer Lateralsklerose, Jahrb. f. Psychiat. u. Neurol. **17**:221, 1898.

12. Dercum, F., and Spiller, W.: A Case of Amyotrophic Lateral Sclerosis Presenting Bulbar Symptoms with Necropsy and Microscopic Examination, J. Nerv. & Ment. Dis. **26**:84, 1899.

13. Czylark, E., and Marburg, O.: Beiträge zur Histologie und Pathogenese der amyotrophischen Lateralsklerose, Ztschr. f. klin. Med. **43**:59, 1901.

14. Mott, F. W., and Tredgold, A. F.: Some Observations on Primary Degeneration of the Motor Tract, Brain **25**:401, 1902.

15. Lejonne, P., and Lhermitte, J.: Un cas de sclérose amyotrophique à forme anormale avec autopsie, Rev. neurol. **14**:485, 1906.

16. Babonneix, L., and Widiez, A.: Sclérose combinée; lésions diffuses et inflammatoires du névraxe, anévrysme de l'artère spinale antérieure, Rev. neurol. **1**:1214, 1930.

17. Dagnélie, J., and Cambier, P.: Contribution anatomo-clinique à l'étude de la sclérose latérale amyotrophique, Rev. neurol. **2**:25, 1933.

18. Digested gastric juice from a patient (A) suspected to have pernicious anemia is given to a patient (B) known to have pernicious anemia, untreated with liver. Improvement in the clinical symptoms and blood picture in B signifies that A has the antianemic factor and therefore does not have pernicious anemia. Lack of improvement in the blood picture of B indicates that A does not have the antianemic factor and therefore has pernicious anemia.

spinal cord changes may precede the anemia and persist for a long time in its absence. Holmes and Stone pointed out the combination of subacute combined degeneration and anterior horn cell disease in pernicious anemia. Syphilis and other infections, as well as arteriosclerosis of the spinal vessels, may produce lesions of the spinal cord simulating amyotrophic lateral sclerosis and at the same time involve the sensory pathways. No such evidence was found in our cases.

SUMMARY

1. Two cases of amyotrophic lateral sclerosis with changes in the sensory pathways are described. These changes consisted in astereognosis, impairment in two point discrimination and loss of sense of position in the fingers of both hands in the first case and in hypesthesia and hypalgesia in the distal portions of the extremities and absence of vibratory sensation in the lower extremities in the second case.

2. Clinically, such cases may have to be differentiated from those of subacute combined degeneration. The Castle test and the presence or absence of free hydrochloric acid are of diagnostic importance.

3. Histopathologically both patients showed, in addition to the typical lesions of amyotrophic lateral sclerosis, degeneration of the posterior columns at one or two levels. In subacute combined degeneration such a combination is generally found throughout the cord.

4. The similarity of the pathologic process in the posterior to that in the lateral columns favors the view that we are dealing with one underlying cause and not a chance association.

ENCEPHALOGRAPHIC STUDIES IN CASES OF EXTRAPYRAMIDAL DISEASE

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We are presenting studies made in a group of cases of extrapyramidal diseases from the encephalographic point of view with an attempt at correlation with the clinical findings.

Although cerebral aerographic studies are of comparatively recent origin, an extensive literature on this subject has already sprung up. The procedure has come to be recognized as a method of determining in vivo the underlying cerebral pathologic process. In a group of cases of extrapyramidal diseases we have attempted to correlate by such methods the clinical symptomatology with the existing cerebral changes. Studies of chronic, degenerative and fixed lesions of the central nervous system have been made by Kruse,[1] Flügel,[2] Walker[3] and others, but we know of no investigation of this particular group as an entity.

Cases with such syndromes have been described, but mainly in children or in patients in whom the existing process was of recent origin. At the Montefiore Hospital we have had the opportunity of following our series of patients over a long period of years so that we are in a position to compare the aerographic findings in cases of disease of long standing with those that have already been reported. In addition, we have been able to visualize, because of the varying duration of the disease in cases in each separate group, cross-sections at different levels in the same disease. We are adding to these observations studies on a group of clinical disease entities which have not as yet been investigated roentgenographically.

From the Neurological Division of the Montefiore Hospital, service of Dr. S. Philip Goodhart.

Read before the Section on Nervous and Mental Diseases at the Joint Meeting of the American Medical Association and the Canadian Medical Association, Atlantic City, N. J., June 12, 1935.

1. Kruse, F.: Cerebrale Krankheiten des Kindesalters in typischen Encephalogrammen, Ergebn. d. inn. Med. u. Kinderh. **37**:333, 1930.

2. Flügel, F. E.: Die Encephalographie als neurologische Untersuchungsmethode, Ergebn. d. inn. Med. u. Kinderh. **44**:327, 1932.

3. Walker, A. E.: Encephalography in Children, Am. J. Roentgenol. **32**:437, 1934.

Our technical procedure throughout this work has been totally to replace the spinal fluid with air through lumbar subarachnoid injection, using the simple syringe method. The patient is given 0.2 Gm. of sodium amytal one hour before, and 0.015 Gm. of morphine sulfate hypodermically fifteen minutes before, the puncture. In all cases except those of Huntington's chorea, satisfactory anesthesia resulted. Antero-posterior and postero-anterior and lateral encephalograms were then made in both the prone and the erect position.

The Stanford modification of the Binet-Simon test was employed in the psychometric examination of these patients. All the psychometric examinations were done in January 1935.

CHRONIC DEGENERATIVE CHOREA (HUNTINGTON'S CHOREA)

We were unable to find any reports in the literature on encephalographic studies in cases of chronic degenerative chorea. We are reporting studies made in three cases. In the first two the condition was more severe than in the third, with the roentgenographic findings compatible with the variation in the clinical pictures.

CASE 1.—M. M., a woman aged 43, was admitted to the hospital on Nov. 16, 1932. The patient's father died of Huntington's chorea at the age of 48. Her dyskinesia started at 34 with prodromal symptoms of insomnia, and the movements have become progressively worse. It was impossible to date the onset of the mental symptoms, but on admission they were already manifest. The mental age was 7 years, and the intelligence quotient, 44.

Encephalograms (fig. 1).—One hundred and eighty cubic centimeters of spinal fluid was removed, and 175 cc. of air was injected. There was moderate bilateral dilatation of both ventricles, the right being greater than the left, involving predominantly the frontal horns and the first third of the bodies of the lateral ventricles. The third and fourth ventricles and the aqueduct were slightly dilated. There was moderately severe cortical atrophy involving the superior temporal, supramarginal, angular, middle and inferior frontal gyri and the island of Reil.

CASE 2.—M. S., a man aged 51, was admitted to the hospital on Nov. 13, 1930. The patient's father died at the age of 40 and a brother at the age of 34 of Huntington's chorea. The dyskinesia started at 40 and became progressively worse. Intellectual impairment was first observed at 45 and has progressed rapidly. Psychometric examination was unsuccessfully attempted on this patient with tests as low as the third and fourth year levels.

Encephalograms (fig. 2).—One hundred and seventy-five cubic centimeters of spinal fluid was removed, and 170 cc. of air was injected. There was symmetrical bilateral ventricular dilatation. The third and fourth ventricles and the aqueduct were slightly dilated. There was moderately severe bilateral cortical atrophy involving the superior, temporal, inferior frontal and inferior portions of the motor area, the supramarginal, angular and inferior parietal convolutions and the island of Reil.

CASE 3.—T. L., a woman aged 34, was admitted to the hospital on Feb. 19, 1935. The patient's father was a patient in this hospital in 1915 with a condition that was diagnosed Huntington's chorea. The patient's dyskinesia started at the

age of 27 and became progressively worse. It was impossible to date the onset of the mental symptoms, but these were present at the time of admission. The mental age was 7 years and 8 months, and the intelligence quotient, 48.

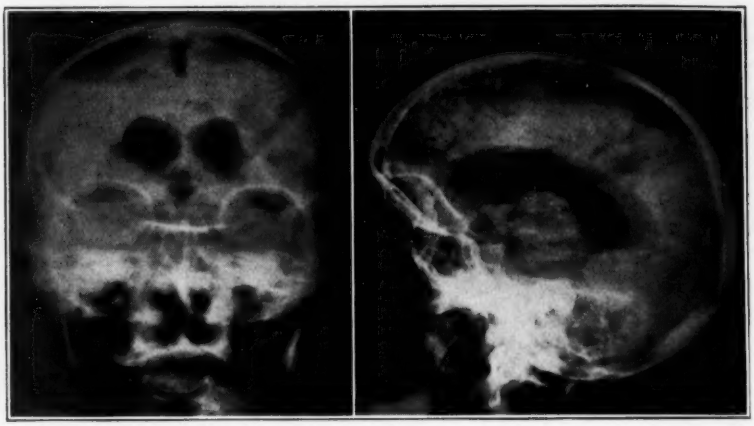

Fig. 1. (case 1).—Anteroposterior and lateral encephalograms, showing moderate bilateral dilatation of both ventricles, the right being greater than the left, involving predominantly the frontal horns and the first third of the bodies of the lateral ventricles. The third and fourth ventricles and the aqueduct are slightly dilated. There is moderately severe bilateral cortical atrophy.

Fig. 2 (case 2).—Anteroposterior and lateral encephalograms, showing symmetrical bilateral ventricular dilatation. The third and fourth ventricles and the aqueduct are slightly dilated. There is moderately severe bilateral cortical atrophy. The condition is more advanced than that shown in figure 1.

Encephalograms (fig. 3).—One hundred and thirty cubic centimeters of spinal fluid was removed, and 125 cc. of air was injected. As in the preceding two cases, there was moderate bilateral internal hydrocephalus, with the frontal horns

and the first third of the bodies of the lateral ventricles more involved than the remainder of the ventricular system. There was moderate cortical atrophy, which was most marked in the right hemisphere.

Comment.—The outstanding features in these three cases were the dilatation of the anterior horns and anterior third of the bodies of the lateral ventricles associated with general cortical atrophy. Dilatation of this segment of the ventricles has been correlated by many authors (Kruse and others) with atrophy of the basal ganglia, more especially the head of the caudate nucleus, and this correlation has become generally accepted. In cases of Huntington's chorea, gross examination of the brain reveals shrinkage of the putamen and caudate nuclei and generalized cortical atrophy, including the island of Reil. Thus, it is evident that aerographic findings correspond with the pathologic changes.

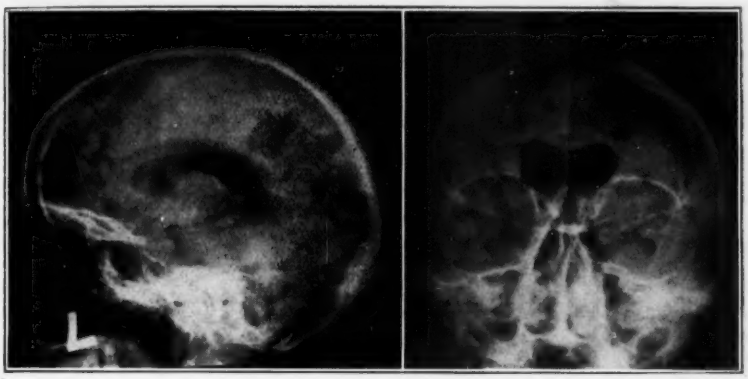

Fig. 3 (case 3).—Anteroposterior and lateral encephalograms. As in the preceding two cases, there is moderate bilateral internal hydrocephalus with the frontal horns and first third of the bodies of the lateral ventricles more involved than the remainder of the ventricular system. There is moderate cortical atrophy, most marked in the right hemisphere.

As in these three patients the condition clinically is uniformly more advanced in respect to the dyskinesia and mental status, so roentgenographically the encephalograms of patients with the most severe manifestations show a uniformly more extensive process. This demonstrates that the disease in Huntington's chorea progresses as a synchronous atrophy of the cerebral structures involved.

PROGRESSIVE HEPATOLENTICULAR DEGENERATION (WILSON'S DISEASE, WESTPHAL-STRÜMPEL'S PSEUDOSCLEROSIS)

Scattered reports of encephalographic studies in cases of progressive hepatolenticular degeneration are found in the literature. Our group

includes three cases, one rather advanced and the other two, relatively recent (in twin sisters).

Case 4.—R. W., a woman aged 19, was admitted to the hospital on Jan. 21, 1932. There was a familial history of a similar disease in two younger sisters, 13 and 15½ years of age, respectively. There was a history of transitory "jaundice" at the age of 7 and migrating polyarthritis at 12 years, of five months' duration. At 14, there developed severe headaches in the right temporal region and drowsiness, lasting for a year and accompanied by impairment of vision. At 15, dysarthria and a tremor of the hands that became exaggerated on intentional movement were first noticed. These symptoms have become progressively worse, and at present the patient has flying movements (*Fluegel-Schlagen*) of both upper extremities. Examination showed a "mechanical doll" gait, dysarthria, gross intention tremors with *Fluegel-Schlagen,* pendulous knee jerks bilaterally, ataxia with cog-wheel rigidity, a Babinski sign on the right, a Kayser-Fleischer ring in each

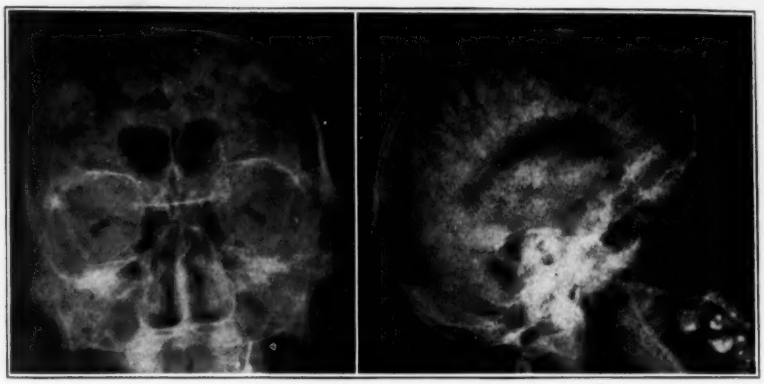

Fig. 4 (case 4).—Anteroposterior and lateral encephalograms. The frontal horns, the anterior portion of the bodies of the lateral ventricles and the fourth ventricle are dilated. There are coarse sulci in the precentral and premotor areas.

eye and acne vulgaris of the face. The mental age was 10 years and 6 months, and the intelligence quotient, 66.

Encephalograms (fig. 4).—One hundred and sixty cubic centimeters of spinal fluid was removed, and 155 cc. of air was injected. The frontal horns and the anterior portion of the bodies of the lateral ventricles and fourth ventricle were dilated. The sulci were coarse in the precentral and premotor areas. The enlargement of the fourth ventricle was thought to be due to cerebellar atrophy.

Case 5.—B. H., a girl aged 16, was admitted to the hospital on Sept. 12, 1934. At the age of 10 the patient had a period of nausea and vomiting which lasted three months. At that time she was operated on at another hospital, where biopsy of a specimen from the liver showed cirrhosis. She was well until 12 years of age, when, following an emotional upset, dysarthria developed. Since November 1934 she has been complaining of frontal headaches. Examination in January 1935 revealed ataxia of all extremities, coarse action tremor and rigidity of the upper extremities, rhythmic tremor and weakness of the tongue, masked facies, facial

acne vulgaris and Kayser-Fleischer rings. The mental age was 11 years, and the intelligence quotient, 75.

Encephalograms (fig. 5).—One hundred and twenty-seven cubic centimeters of spinal fluid was removed, and 120 cc. of air was injected. The anterior portions of both lateral ventricles were dilated, but no other abnormalities were present.

CASE 6.—E. H., the nonidentical twin sister of the patient in case 5, was admitted to the hospital on Sept. 12, 1934. Bitemporal headaches were present from the age of 6 to the age of 13. Tremors began in the hands at 14½ and had since involved the lower limbs. There were no complaints referable to the gastrointestinal tract. Examination in January 1935 showed rhythmic tremors of medium amplitude involving all the extremities and the tongue, slight ataxia, weakness and early fatigue of both upper extremities, the right being greater than the left, cog-wheel rigidity of all extremities, masked facies, facial acne vulgaris and Kayser-Fleischer rings. The mental age was 13 years and 8 months, and the intelligence quotient, 85.

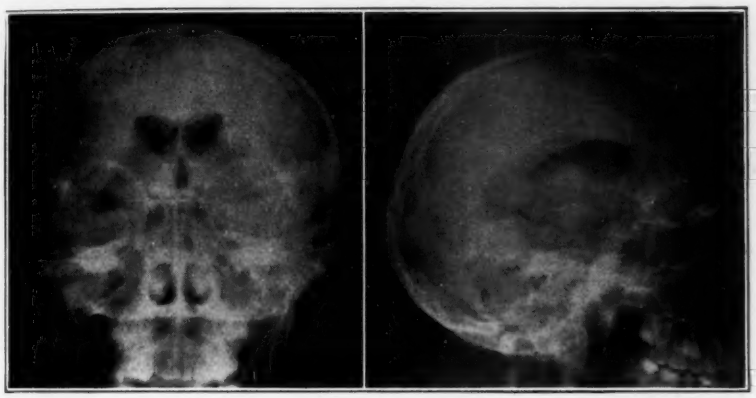

Fig. 5 (case 5).—Anteroposterior and lateral encephalograms. The anterior horns and the first third of both lateral ventricles are dilated. Otherwise this picture is normal.

Comment.—Three cases of this syndrome in various stages of development have been presented; in the case with the shortest clinical history there was merely a slight dilatation of the anterior horns of the lateral ventricles; in the case in which the disease was next most advanced there was more dilatation of the anterior horns. In neither case, however, was there any encephalographic evidence of other involvement. In the case of most advanced disease encephalographic changes were noted not only in the ventricular system but in other structures. This suggests that in contrast to Huntington's chorea, in which the involvement, even in the case of an earlier stage, was uniform throughout the brain, in Wilson's disease the pathologic process tends to advance more rapidly in the basal ganglia than in the other areas which are involved at a later time. We realize that aerographic studies

can in no way tell what finer changes may be taking place; however, the chronological appearance of the clinical symptoms tends to confirm such implications.

It is well known that in cases of Huntington's chorea the mental symptoms not only develop with dyskinesia but frequently actually precede it, so that cortical atrophy may be expected at an early date. In Wilson's disease, on the other hand, dyskinesia always precedes the appearance of the mental symptoms. This is in accord with the absence of demonstrable cortical changes in the earlier stages.

The preponderance of involvement of the basal ganglia as revealed at postmortem examination is in accord with these principles. In addition, there is noted at necropsy involvement of the cortical structures and the dentate nuclei and occasionally of the pons. The roentgenologic

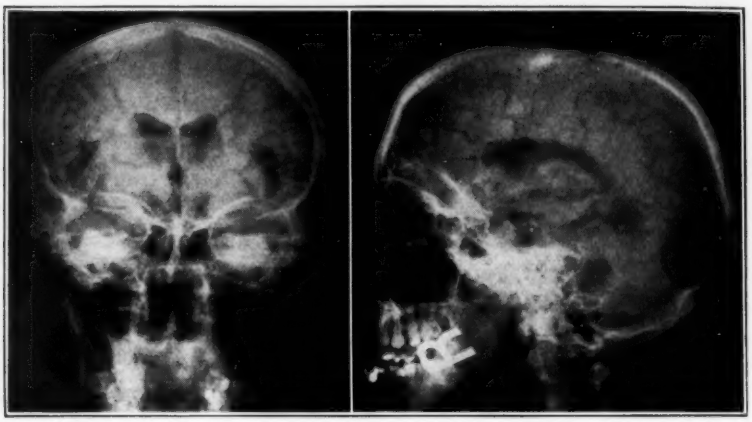

Fig. 6 (case 6).—Anteroposterior and lateral encephalograms. The anterior horns and the first third of both lateral ventricles are dilated. Otherwise the picture is normal.

picture in our case of advanced involvement shows these first-named structures to be involved.

DYSTONIA MUSCULORUM DEFORMANS

We report the encephalographic studies in three cases of the dystonic type of dyskinesia. The first two are cases of iodiopathic dystonia musculorum deformans; the third is a case of symptomatic dystonia in a patient with diffuse involvement of the central nervous system, possibly posttraumatic.

Case 7.—J. S., a youth aged 17, was admitted to the hospital on Jan. 6, 1935. At 9 years of age dystonic movements of the right arm began and were followed by similar movements in the right sternocleidomastoid muscle, the left arm, the left sternocleidomastoid muscle and the long muscles of the back. Examination

in January 1935 showed tortipelvis, torticollis and dystonic movements of both upper extremities. There was a dromedary gait. The mental age was 16 years, and the intelligence quotient, 100.

Encephalograms (fig. 7).—One hundred and sixty cubic centimeters of spinal fluid was removed, and 150 cc. of air was injected. Except for slight dilatation of the third ventricle, there were no abnormal findings.

CASE 8.—M. B., a boy aged 14, was admitted to the hospital on Feb. 26, 1935. There was a history of jerky movements of the head since the age of 4 years. These progressed to involve the right leg and hip. The patient started to school at 6 and at the time of admission was enrolled in the first term of high school. Examination in January 1935 showed torticollis and tortipelvis, with dystonic movements of the head, the right arm and the right leg. There was a right dorsal scoliosis, and the right shoulder was higher than the left. The patient walked with a dromedary gait and had dysarthria. The mental age was 13 years and 7 months, and the intelligence quotient, 92.

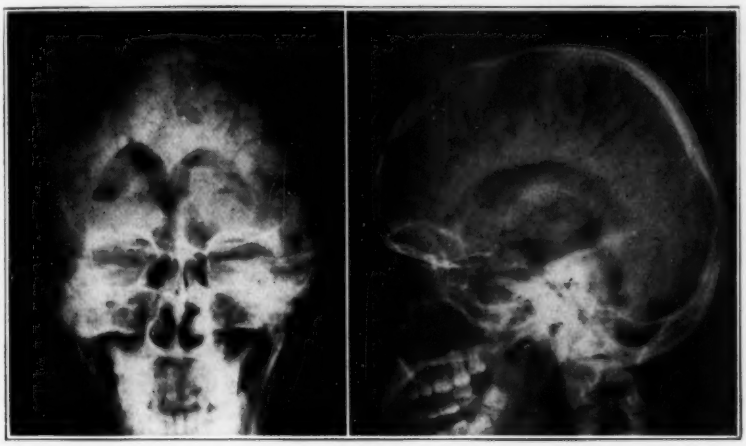

Fig. 7 (case 7).—Anteroposterior and lateral encephalograms. Except for slight dilatation of the third ventricle, no abnormal findings were noted.

Encephalograms (fig. 8).—One hundred and fifteen cubic centimeters of spinal fluid was removed, and 110 cc. of air was injected. No abnormalities were observed.

CASE 9.—H. R., a man aged 43, was admitted to the hospital on April 7, 1919. At the age of 20 he had met with an accident accompanied by unconsciousness. Four months later there developed dyskinetic movements in the muscles of the hamstring group of the left thigh followed by similar involvement of both upper extremities and the left side of the face. Examination in January 1935 revealed rigidity of the facial muscles resulting in a persistent risus sardonicus, deviation of the tongue to the left, postural design of "main en griffe" in both hands, atrophy of the adductor muscles of the left thigh and dystonia of the feet. There were generalized hyperreflexia and hypertonia. Because of the last-mentioned symptoms, movements of all muscle groups were restricted. There was gradual but steady mental deterioration during the patient's fifteen years of hospitalization. In January 1935 the mental age was 10 years, and the intelligence quotient, 63.

Encephalograms (fig. 9).—Three hundred and twenty cubic centimeters of spinal fluid was removed, and 300 cc. of air was injected. There was generalized cortical atrophy, with coarse sulci, most marked in the postcentral and parietal regions, and enlargement of the cisterna ambiens. The ventricular system was not dilated. Air was seen under the tentorium, and there was some cerebellar atrophy.

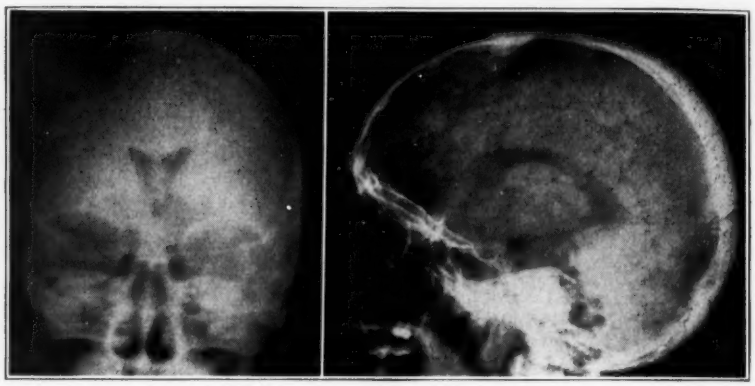

Fig. 8 (case 8).—Anteroposterior and lateral encephalograms. No abnormalities were seen.

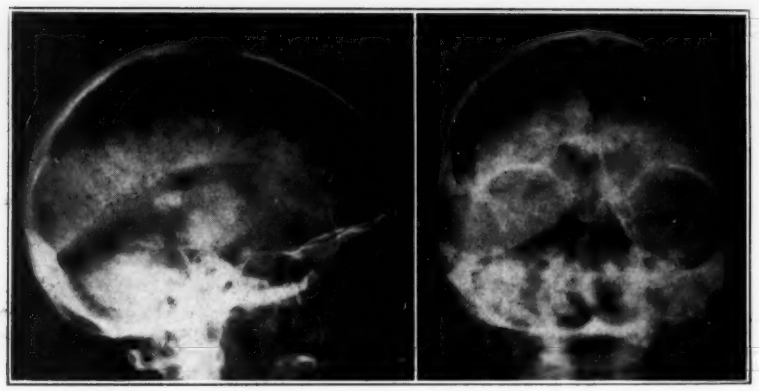

Fig. 9 (case 9).—Anteroposterior and lateral encephalograms. There are generalized cortical atrophy with coarse sulci, which is most marked in the postcentral and parietal regions, and dilatation of the cisterna ambiens. Air is seen under the tentorium, and there is some cerebellar atrophy.

Comment.—The concepts of the pathologic process of this disease are controversial. Two patients with this disease came to necropsy at our hospital, and there was no gross pathologic change. The absence of aerographic abnormalities in the two cases of idiopathic dystonia

musculorum deformans reported here is in accord with these pathologic studies. It is obvious that in the symptomatic group, the pathologic process will vary in each case so that there can be no uniformity in the aerograms. In our case there was a correlation between the diffuse clinical involvement and the extensive encephalographic changes.

We have insufficient material from which to draw conclusions, but the possibility presents itself that the encephalogram may be a means of differentiating idiopathic from symptomatic dystonia.

DOUBLE ATHETOSIS—LITTLE'S DISEASE

Under the classification of Little's disease we include cerebral palsy in children which is due to a lesion which is either congenital or acquired during birth or early childhood. From this heterologous group an essen-

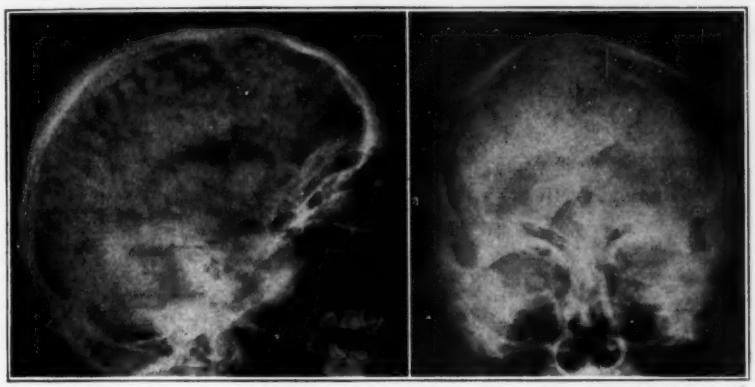

Fig. 10 (case 10).—Anteroposterior and lateral encephalograms. No abnormalities were seen.

tially extrapyramidal entity, double athetosis, may be differentiated. Of a group of four cases of double athetosis which we have studied aerographically, we detail the findings in one typical case.

CASE 10.—G. H., a man aged 36 was admitted to the hospital on March 30, 1919. The family history was irrelevant. The patient was a full term, spontaneously delivered child. He was never able to walk and could utter only a few words at 2 years of age. At 8 he had convulsions which occurred once weekly for about six months, but he had had none since. Examination in January 1935 showed inability to walk, wasting of the muscles of all extremities, continuous athetotic movements in the fingers and toes, generalized hypertonus and hyperreflexia, cerebellar dyssynergia, facial grimacing and explosive speech. These neurologic findings are identical with those described in the notes on admission.

Encephalograms (fig. 10).—One hundred and seventy-three cubic centimeters of spinal fluid was removed, and 168 cc. of air was injected. The encephalogram revealed no abnormalities.

Comment.—In our group of four cases the encephalographic findings were within normal limits despite the severity of the disease. This is compatible with the pathologic lesions in this condition as described by Dunlap,[4] Davison [5] and others, namely, status marmoratus and status dysmyelinisatus. In both status marmoratus and status dysmyelinisatus the pathologic process is of such nature that there is no gross distortion in the configuration of the ventricular structures. Thus, a priori, one would expect no aerographic changes. The encephalogram, therefore, offers an additional means for establishing the specific entity of double athetosis and of differentiating this group from cerebral infantile palsy with dyskinesia in which the latter is merely a secondary superimposition.

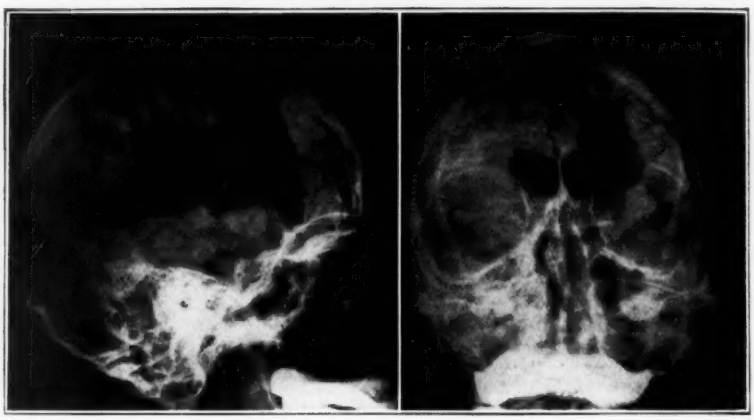

Fig. 11 (case 11).—Anteroposterior and lateral encephalograms. There are bilateral internal hydrocephalus and cortical atrophy, more marked on the right, with an area of porencephaly measuring 2.8 by 4 by 7 cm.

We report one of the more classic types of Little's disease from an encephalographic investigation of five cases.

Case 11.—S. C., a woman aged 31, was admitted to the hospital on April 11, 1913. The patient had been prematurely delivered at 7 months, weighing 1½ pounds (680.38 Gm.) at birth. At 5 months clumsiness of the left arm was first noticed. Dentition started at 11 months. The patient walked with support at 3 years, but never alone. At 2 years of age there developed flexor spasms of the left upper and both lower extremities, which were more marked on the left, with

4. Dunlap, C. B.: Structural Changes in Huntington's Chorea, Brain **50:** 631, 1927.

5. Davison, Charles: Diseases of the Extrapyramidal System, in Blumer, G.: The Practitioner's Library of Medicine and Surgery, New York, D. Appleton-Century Company, Inc., 1936, vol. 9, chap. 29.

bilateral equinovarus. Examination on admission showed spastic paresis of the left upper and both lower extremities, which was most marked in the left lower extremity, with hyperreflexia in these limbs and a bilateral Babinski sign. There was some degree of atrophy in the affected extremities. The right upper extremity was spared. The neurologic status remained practically unchanged during the patient's twenty-two years in the hospital.

Encephalograms (fig. 11).—One hundred and eighty cubic centimeters of spinal fluid was removed, and 175 cc. of air was injected. There was a large area of porencephaly in the right frontal area measuring 2.8 by 4 by 7 cm. There was bilateral internal hydrocephalus which was more marked on the right. The third and fourth ventricles and the aqueduct were not dilated. The cisterna pontis was increased in size.

Comment.—In our group of cases, in patients whose ages varied from 19 to 41 the encephalographic findings were similar to those reported by previous authors in children. This would suggest that the lesions in these cases were fixed, as would also the lack of clinical progress in the case described here. There were no typical encephalograms, as would be expected in such a heterologous clinical group. In the aerograms reported on internal hydrocephalus, cortical atrophy and porencephaly as well as normal findings have been noted. Correlation between the encephalographic and the clinical findings was possible in case 11, in which the predominant clinical signs were contralateral to the large area of porencephaly demonstrated.

SUMMARY

A group of cases of extrapyramidal disease were studied encephalographically, and the following observations were made:

The encephalograms in three cases of Huntington's chorea revealed ventricular dilatation, predominantly of the anterior portions of the lateral ventricles, plus cortical atrophy.

The encephalograms in three cases of Wilson's disease, two of comparatively early and one of advanced involvement, disclosed dilatation of the anterior portion of the lateral ventricles in the former and cortical atrophy accompanied the ventricular dilatation in the latter.

The encephalograms in three cases of dystonia, two of the idiopathic and one of the symptomatic type, were normal in the former and disclosed diffuse involvement in the latter.

The encephalograms in four cases of double athetosis were normal.

The encephalograms in five cases of Little's disease revealed porencephaly in two instances and internal and external hydrocephalus in the remaining three cases.

ABSTRACT OF DISCUSSION

Dr. A. J. Bendick, New York: I want to show a new technic that my associates and I are developing and have used in the study of extrapyramidal disease. So far as we could find out in the literature fluoroscopic studies have never been

made in cases of such disease. It is surprising how clearly one can see the air in the ventricular system when this method is used. We put our patient on the tilt table. When we have air in the part of the ventricular system that we desire to outline the picture is taken.

Dr. Charles Davison, New York: Dr. Goodhart and his associates have given an excellent demonstration of encephalographic studies in cases of extrapyramidal disease. In a case of Huntington's chorea there were marked shrinkage of the caudate and putamen and dilatation of the anterior horns of the lateral ventricles. The ventricular dilatation corresponds to that shown by Dr. Goodhart and his associates in the roentgenograms.

In cases of progressive hepatolenticular degeneration a twofold process may occur. When the striatum and other diencephalic structures are not markedly shrunken, the ventricular system appears normal. In case of bilateral destruction of the lenticular nuclei, the anterior horns of the lateral ventricles are dilated.

I had the opportunity to make studies in two cases of degenerative musculorum deformans in which necropsy was performed. Grossly, in neither of these cases was there marked destruction of the striatum or pallidum. However, changes were demonstrated microscopically. The ventricular system, as in the cases of the degenerative type reported by the authors, was normal. In the cases of symptomatic dystonia with more marked destruction of the basal ganglia, one should expect dilatation of the ventricular system.

There are three types of cases of cerebral diplegia: (1) the so-called status dysmyelinisatus, with paling of the pallidal structures without any alterations in the ventricular system; (2) the status marmoratus, in which the striatum and pallidum have a marble appearance without any changes in the ventricular system, and (3) Little's disease, in which hemorrhages and porencephalic defects may be found. In the latter there are dilatation of the ventricular system and porencephaly.

Dr. Goodhart and his associates, by these encephalographic studies, were able to foretell the pathologic entity. This method is especially of value in differentiating the various forms of cerebral diplegia.

Dr. Tracy Putnam, Boston: It seems to me that this is an important method of approaching the question of the disorders of movement, and particularly important since there is now an operative treatment that is effective against some of them.

My associates and I have carried out encephalography in all our cases of athetosis. They have fallen into a slightly different group from most of those that Dr. Goodhart and his associates have presented. In most of them the condition has been due to trauma at birth or to trauma in adult life. They have been mainly instances of unilateral athetosis, and we believe that we have observed a quite characteristic encephalographic finding, an apparent atrophy of the caudate nucleus with dilatation of the ventricle downward. This has regularly been present, although sometimes, at least, in association with other defects, such as cortical atrophy or more marked destruction of the basal nuclei. It is a valuable method of distinguishing between the various types of extrapyramidal disease.

Dr. S. P. Goodhart, New York: I suppose that the cases that Dr. Putnam has indicated were not those described as instances of double athetosis. His method appears to be a satisfactory one.

AN ELECTROMYOGRAPHIC STUDY OF MYOTONIA

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Myotonia, the persistent contraction of a muscle after voluntary, mechanical, electrical or, in some instances, reflex stimulation, is the chief symptom of myotonia congenita, or Thomsen's disease. Myotonia is also a prominent symptom of certain other muscular dystrophies, particularly dystrophia myotonica (myotonia atrophica).

Erb, in 1886, described the essential clinical and pathologic manifestations of myotonia congenita as well as the characteristic reactions of myotonic muscles to electrical stimulation. Curschmann [1] was one of the first to take into full account the extramuscular symptoms which may be associated with myotonia and atrophy in dystrophia myotonica. Since summaries of the literature and reviews of some of the more typical cases reported may be found in publications by Jacoby,[2] Johnson and Marshall,[3] Rosett,[4] Adie and Greenfield,[5] Jelliffe and Ziegler [6] and others, no attempt will be made here to repeat the detailed descriptions of these related syndromes. Suffice it to say, as Adie and Greenfield [5] have pointed out, that the myotonia of myotonia congenita and that of dystrophia myotonica are identical. The following report of clinical and electromyographic examinations, which were made in a case of myotonia congenita and one of dystrophia myotonica, support this contention.

Numerous theories have been proposed to account for the phenomena observed in myotonic muscles, but none seems to offer a satisfactory explanation. The present study was undertaken with the hope that newer and more refined methods of electromyographic study might make it possible to learn more about the mechanism of this disorder.

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* National Research Fellow.

From the Departments of Physiology and Neuropathology, Harvard Medical School, and the Neurological Service of the Massachusetts General Hospital.

1. Curschmann, H.: Deutsche Ztschr. f. Nervenh. **45**:161, 1912.
2. Jacoby, G. W.: J. Nerv. & Ment. Dis. **25**:509, 1898.
3. Johnson, W., and Marshall, G.: Quart. J. Med. **8**:114, 1915.
4. Rosett, J.: Brain **45**:1, 1922.
5. Adie, W. J., and Greenfield, J. G.: Brain **46**:73, 1923.
6. Jelliffe, S. E., and Ziegler, L.: Thomsen's Disease (Myotonia Congenita): Report of Case and Review of American Literature, J. A. M. A. **100**:555 (Feb. 25) 1933.

REPORT OF CASES

CASE 1.—*Myotonia congenita.*

J. M., an American boy of Italian extraction, aged 18, was first observed in the neurologic outpatient department of the Massachusetts General Hospital in October 1933. He complained that on sudden strong voluntary contraction cramp-like tightening of the muscles occurred and that release could not be immediately effected. The condition had been present as long as he could remember and was particularly manifest in the act of shaking hands, in which his grip appeared normal and firm but was abnormally prolonged. On suddenly rising from a sitting position or on starting to run, his legs would become cramped, and he would topple over. Apparently, delayed relaxation in the muscles first brought into sudden use did not permit the normal reciprocal action of antagonists necessary for coordinated movement.

The degree of myotonia was roughly proportional to the intensity of the contraction, and only strong or moderately strong contractions produced noticeable myotonic reactions. The myotonia was enhanced by cold, damp weather, fatigue and emotional excitement. Preliminary activity or "warming up" decreased the myotonic effects. The patient was sensitive about his condition but had adjusted himself to it adequately. He had learned not to make quick movements without first limbering up, and he gripped only lightly on shaking hands, so that he was able to conceal his defect from the casual observer. He was sociable, cooperative and alert and displayed no unusual mental reactions.

The family history revealed little definite evidence of myotonia or psychic symptoms, sometimes reported as related hereditary factors. The father, a laborer, was in good health and free from myotonia. Several members of the father's family, including his mother, appeared to be in good health and without myotonic symptoms. The patient's mother described a "cramp" she had once experienced in the muscles of her hand, but her reports were inadequate and not reliable. She also reported vague "nervous" manifestations and appeared to be of a neurotic constitution. Two siblings, both girls, aged 5 and 16, revealed no evidence of myotonia but showed marked development of the muscles of the limbs, similar to that of the patient.

Physical examination revealed that the patient was well developed, of athletic build and 5 feet, 9 inches (175 cm.) in height and weighed 155 pounds (7.3 Kg.). His muscles were firm and symmetrical, appearing almost hypertrophic (fig. 1). His posture was careless, and his gait suggested spasticity until he was "warmed up." There were no abnormalities of the head, throat, heart, lungs or abdomen and no evidence of testicular atrophy. He complained of moderate constipation.

The senses of smell and taste, vision and hearing were unimpaired. The ocular fundi were normal, and there was no diplopia, nystagmus, strabismus, lid-lag or exophthalmos. The pupils reacted to light, but there was a slight delay in accommodation, and the extrinsic muscles of the eyes were involved in the myotonia. The motor elements of the fifth and seventh cranial nerves also were involved, but there was no weakness or paralysis. There was a sensation of stiffness in the facial muscles on chewing, laughing or smiling, and a slight delay in relaxation was sometimes observed. There was slight asymmetry of the mouth and naso-labial folds but no deviation of the jaw. The tongue was involved in the myotonia, and there was slight transient difficulty in swallowing and in articulation. Protrusion of the tongue was not hampered, but movements from side to side were difficult until the muscles had been "warmed up." The sternocleidomastoid and trapezius muscles showed marked myotonic involvement.

There were no convulsions, twitchings, tremors or paralyses and no ataxia or true spasticity. The tendon reflexes were normal; the abdominal reflexes were present and active. There was no true adiadokokinesis, and after a "warming up" the movements were smooth and free as long as continued. Normal responses were obtained when forced grasping, the fanning sign and Rossolimo's tendon reflex were looked for. The Romberg, Babinski, Chvostek and Trousseau signs were not present.

All the voluntary muscles of the body were involved in the myotonic disorder, but the flexor muscles in the forearms and the extensor muscles in the thighs

Fig. 1.—Myotonia congenita. The muscles appear almost hypertrophic and all show myotonic reactions.

were more prominently involved. Involvement was not limited to voluntary activity, for reflex coughing and sneezing produced contractions which were released slowly in the rectus abdominis and latissimus dorsi muscles. In response to mechanical percussion with a reflex hammer, linear grooves formed along the lines of muscle cleavage and disappeared slowly as the muscle gradually relaxed. After-contraction persisted for approximately three seconds in all muscles after strong voluntary, mechanical or electrical stimulation. The strength of the grip was not abnormally weak but was less than one might expect.

Several representative flexor and extensor muscles of both limbs were observed in response to stimulation with faradic and galvanic currents. The normal twitch

was not obtained on stimulation with galvanic current; instead a persistent tetanic contraction was observed as long as the current was flowing. If stimulation was strong the contraction persisted for about three seconds after the current ceased to flow. The same prolonged after-contraction was observed following strong faradic stimulation. The strength-duration curve resulting from chronaxie determinations on the flexor carpi radialis muscle was within normal limits. The chronaxie was 0.093 milliseconds, which is within the range of variation for normal subjects.

No biopsy was performed. The results of examination of the blood were within normal limits: The calcium content was 10.6 mg., and the magnesium content was 2.6 mg., per hundred cubic centimeters, and there was 0.23 unit of phosphatase. The Wassermann reaction was negative.

CASE 2.—*Dystrophia myotonica.*

L. H., an American man of Scotch-Irish descent, aged 26, was admitted to the neurologic ward of the Massachusetts General Hospital in November 1934. Four years before he had noticed weakness of the fingers and slight difficulty in swallowing. Coincidental with that he noticed an inability to release his grasp on objects. He also observed that his hands and face often appeared blue and cold after he had been swimming. Weakness of the third, fourth and fifth fingers of each hand, which was due to marked atrophy of the respective flexor muscles of the forearm, had become progressively worse. The inability to release certain muscles after a strong contraction likewise had become more marked. Dysphagia was most noticeable with solid foods, but if he tried to swallow fluids rapidly regurgitation through the nasal passages often occurred. He sweated profusely, and the eyes lacrimated easily.

His father, a lumberman, was killed when a tree fell on him. His mother died two months later of tuberculosis, when the patient was about 6 years of age. An older sister was alive and well. She confirmed the patient's statements that neither she nor the father or mother had any semblance of a similar disorder. The sister had strabismus, and a paternal aunt and uncle both had had operations for the removal of cataracts.

The patient as a child had measles, typhoid and influenza. The tonsils and adenoids were removed at an early age. He occasionally had frontal headaches, which usually appeared in the afternoon. Five years before admission he noticed that the movies hurt his eyes so that he would have to leave the theater. He saw double when objects were close to him. All his teeth were removed six years before because of disintegration. His appetite was good. He had slight constipation. Sexual desire was present, and there was normal potency.

Physical examination revealed that the patient was poorly developed, poorly nourished and lethargic. He was 5 feet and 3 inches (160 cm.) in height and weighed 121 pounds (54.9 Kg.). During the last four years he had lost approximately 14 pounds (6.4 Kg.). His chest was flat, but expansion was fairly good. Roentgenograms of the chest revealed no abnormalities.

The patient was sociable and cooperative and of low average intelligence. There was no adiadokokinesis, intention tremor or dysmetria. The Romberg and Babinski signs were negative, but there was a positive Chvostek sign. He had a steppage gait and slight foot drop as a result of partial atrophy of the dorsiflexor muscles. The olfactory sense was diminished, as the result of a chronic cold in the head. The ocular fundi were normal, and the pupils reacted well to light and in accommodation. There were slight ptosis of both lids and right internal strabismus. Examination with a slit lamp revealed no sign of cataract or other

abnormal changes of the crystalline lens, although a strand of persistent pupillary membrane was present. All the facial muscles were weak and atrophied, resulting in marked facies myopathica (fig. 2). There was palatal weakness on the right side, with deviation of the uvula, but the gag reflex was present. The voice was weak, and articulation was slurred. The sternocleidomastoid muscles were very weak, only a thin strand of fibers remaining on each side. The tongue was smooth and atrophied but moved normally in all directions. There were atrophy and weakness of the triceps muscles, but the biceps muscles were hypertrophic and knotlike when contracted and were possessed of normal strength. There was

Fig. 2.—Dystrophia myotonica. The muscles of the face, neck and forearms show marked atrophy. The biceps, deltoid and gastrocnemius muscles appear hypertrophic and give myotonic reactions.

marked atrophy of the flexor and extensor muscles of the forearms but very little in the thenar and hypothenar muscles. The legs showed normal strength, except in dorsiflexion and inversion.

There were no spasticity, fibrillary twitchings or tremors and no sensory abnormalities. The tendon reflexes of the upper and lower limbs were diminished; the abdominal reflexes were brisk and equal. The hands, face and neck were blue and cold as compared with the rest of the body. Sweating was marked.

Active myotonia was most prominent in the flexor muscles of the first and second fingers, the muscles of the hands, the biceps and the gastrocnemius muscles,

although mechanical and electrical stimulation elicited myotonic reactions in the deltoid, pectoralis major and other muscles not involved by atrophy. Apparently those muscles most hypertrophic in appearance were most involved in the myotonia. Electrical reactions to galvanic and faradic currents were observed in several muscles. All of them maintained a persistent tetanic contraction as long as the galvanic current was flowing. With either a strong galvanic or a faradic current an after-contraction persisted for from five to twenty seconds after the current ceased. Mechanical percussion with a reflex hammer produced an after-contraction which lasted for from five to ten seconds.

Chemical studies revealed nothing abnormal: the calcium content of the blood was 9.94 mg., and the phosphorus content, 4.2 mg., per hundred cubic centimeters. The hemoglobin value was 70 per cent; the cell count, normal, and the Hinton test, negative. A twenty-four hour specimen of urine showed: creatinine, 647 mg., and creatine, none.

A biopsy report, by courtesy of Dr. C. S. Kubik, indicated that tissue from the left brachioradialis muscle, which was considerably atrophied, showed a pronounced variation in the size of the fibers, the diameters ranging from 16 to 100 microns. The fibers were rounded, and there was an increase in the number of nuclei in the sarcolemma, with some "central" nuclei observed. Longitudinal striation was prominent, apparently owing to the spreading apart of fibrils. Tissue from the deltoid muscle, which was not atrophied and not markedly involved in the myotonia, showed, on the other hand, very little increase in the nuclei and sarcolemma and did not differ greatly from normal muscle. The diameters of the fibers ranged from 50 to 60 microns. Longitudinal striation was not prominent, and no "central" nuclei were observed.

EXPERIMENTAL INVESTIGATION

Electromyographic records were obtained from several myotonic muscles of our patients and compared with records similarly obtained from the same muscles of a normal subject. For some of our observations action currents were obtained from a whole muscle group by means of gross pad electrodes placed over the skin, whereas for others we employed a method devised by Adrian and Bronk[7] for studying the electrical response of single motor units. In the latter technic coaxial needle electrodes were used, a steel hypodermic needle acting as a ground lead and a fine insulated wire cemented into its lumen serving as a grid lead to the amplifier. Needle electrodes of this type when inserted into a muscle are capable of isolating the electrical response of a group of muscle fibers innervated by a single motor neuron. When gross pad electrodes were used, they consisted of brass plates (1 by 4 and 4 by 4 cm.) covered with canton flannel and soaked in a saturated solution of sodium chloride. The small one was placed over the approximate motor point of the muscle group to be studied and served as the grid lead to the amplifier. The large electrode was placed over inactive tissue some distance away and served as a ground lead.

The action potentials in all cases were led off to a six stage transformer-coupled amplifier[8] and a Du Bois oscillograph, the oscillations being photographed on bromide paper by means of reflected light.

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7. Adrian, E. D., and Bronk, D. W.: J. Physiol. **67**:119, 1929.

8. Such an amplifier obviously does not give a true picture of the wave form of an action potential, but it does not interfere with the identification of single motor units and the study of their rhythm or the degree of electrical activity in a muscle at any time.

With gross pad electrodes over the flexor muscles of the right forearm, the subject was instructed to clench his fist as strongly as possible at the sound of a signal, which made the initial deviation in the line at the bottom of the record. He was instructed to maintain the contraction until a second signal indicated that he was to relax and open his hand.

Figure 3 *A* is a typical electromyogram obtained from the flexor muscles of the right forearm of a normal subject, and figure 3 *B* is a corresponding record obtained from the same muscle group of a myotonic patient (case 1). In comparing these records it may be noted that the length of time (auditory reaction time) intervening between the initial signal and the first oscillations of the electrical record is very nearly the same in the two records. In a number of other instances the auditory reaction time of both patients has been within the normal range. The action currents recorded from the patient seem to develop in much the same manner as those from the normal subject, although their initial maximum may be achieved less rapidly. One notes also that the amplitude of the action currents recorded from the patient is at first greater and better maintained than in the record from the normal subject but that the level of the amplitude during the

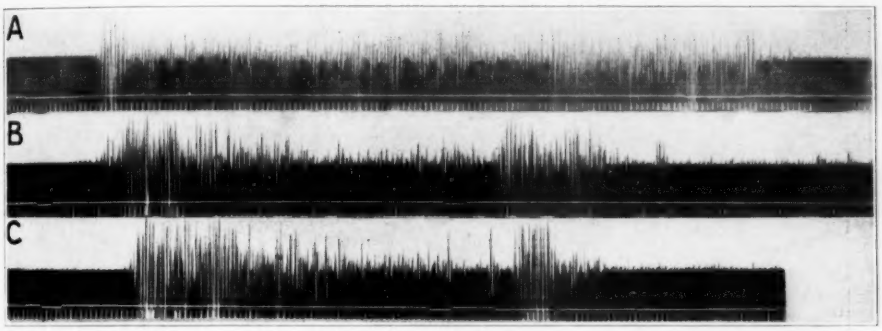

Fig. 3.—Electromyograms from muscles of the forearm. The first deviation in the line at the bottom of each record indicates when the signal was given to clench the fist; the second deviation indicates when the signal for release was given. *A*, normal subject. The action currents subside with the cessation of the voluntary effort. *B*, patient with myotonia congenita. The action currents persist throughout the period of attempted relaxation. *C*, patient with myotonia congenita after "warming up." The time unit equals 20 milliseconds.

maintained contraction falls more in the former than in the latter. These differences may well be attributed to the amount and distribution of energy applied by each subject during the contraction. The most significant difference seems to be in the character of the response recorded after the signal to open the clenched fist has been given. A reaction time after the second signal comparable to that which can be measured after the first signal being allowed, it is noted that the action currents of the normal subject fall off abruptly, whereas in the patient's record two large groups of responses occur during the initial stages of relaxation and a considerable degree of electrical activity persists to the end of the record.

In order to determine the effect of "warming up" on the electrical activity of the muscle during the after-contraction, the patient was allowed to open and close the fist twenty times before making the record shown in figure 3 *C*. This record shows that the electrical activity during the period of relaxation is less

than that shown in figure 3 *B*, prior to the making of which no preliminary "warming up" was permitted.

In the recording of figures 4 and 5 coaxial needle electrodes were employed, the needle being inserted into the right flexor digitorum sublimis muscle, which is largely responsible for flexion of the middle and ring fingers. The middle finger was attached to a torsion wire myograph (mechanograph), the shadow of the lever falling on the film in such a way that its upward swing indicated increasing tension. Thus the curve of the contraction and the electrical activity associated with its production can be observed on the same record.

During the making of the record shown in figure 4 the subject flexed the middle finger on hearing the first auditory signal and attempted to release it immediately after hearing the second signal. Figures 4 *A* and *B* show records for a normal subject, and figure 4 *C*, for a patient with myotonia congenita. Again, as in figure 3, differences may be noted between the patient's record and those of the normal subject. The myograph curve does not rise so steeply during

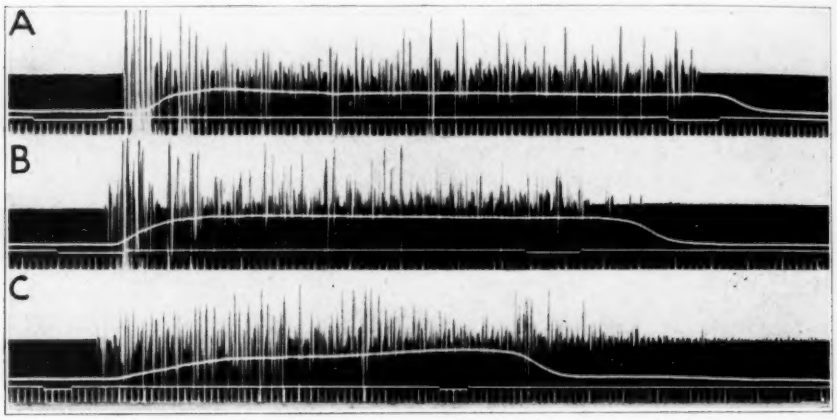

Fig. 4.—Action potentials obtained with needle electrodes inserted in the flexor digitorum sublimis muscle with the middle finger attached to the myograph. Contraction was made on signal and released on signal. *A* and *B*, normal subject showing prompt cessation of action currents and a quick, smooth descent of the curve after the signal to relax. *C*, patient with myotonia congenita, showing persistent action currents during the attempt to relax. The curve does not reach the base level until the end of the record. The time unit equals 20 milliseconds.

the intial phase of contraction in the patient's record. After the usual reaction time following the second signal the action currents of the records for the normal subject cease abruptly, and the myograph curve makes a quick, smooth descent to its base level. In the patient's record the action currents continue throughout the period of attempted relaxation and have not ceased at the end of the record. Also there is a large group of responses accompanying the initial downward swing of the myograph curve. Close examination shows that the descent of the myograph curve is more gradual and of longer duration in the patient's record. The myotonic reaction in this case has a duration of only about one second, but the contraction was not a strong one.

The records shown in figure 5 represent attempts on the part of the patient to relax after a contraction maintained at the level indicated by the myograph record. Since the contraction was moderately strong, responses were recorded from several different motor units, so that it is impossible to follow the response of any single unit. One notes, particularly in records *A* and *B*, that the myograph curve descends smoothly and quickly during the initial stages of relaxation but is suddenly halted and thereafter descends by steplike gradations and reaches a zero or the base level only after more than half a second. The major portion of the action currents may be observed to cease abruptly when the myograph curve starts downward, but a few units were still active, as may be seen in the electrical records of *B* and *C*. There were undoubtedly also active units at the point where the myograph curve starts down in *A*, although they were not in the immediate neighborhood of the electrodes and hence were not recorded. It appears that the persistent activity of these units, although many of those previously active have stopped with the cessation of voluntary effort, maintains a certain amount of tension in the muscle which is responsible for the plateau-like level of

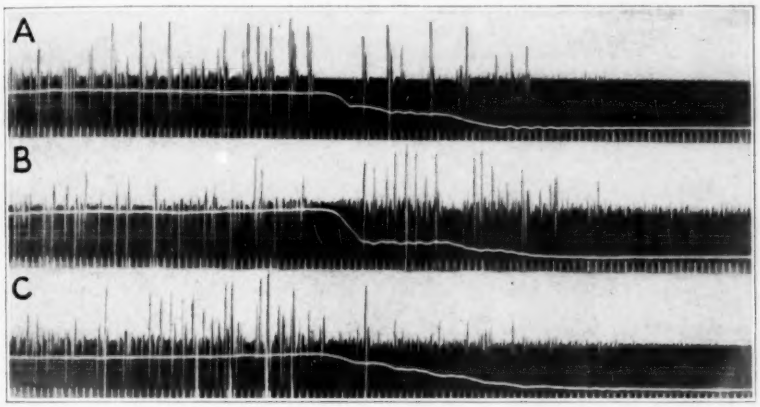

Fig. 5.—The needle electrodes are inserted into the flexor digitorum sublimis muscle of the patient with myotonia congenita while he is maintaining tension against the myograph with the middle finger and suddenly trying to relax it. The curve descends by steplike gradations, and action potentials appear throughout the period of attempted relaxation. The time unit equals 20 milliseconds.

the myograph curve following the first steep descent. Accompanying the plateaus of the myograph curve are marked increases in the electrical activity, which seem to be of reflex character, since they are so definitely correlated with the steplike relaxation or lengthening of the muscle.

In the records thus far presented it is obvious that in the myotonic patients action currents accompany the after-contraction or delayed relaxation phase following the cessation of voluntary effort. This can mean only that the residual contraction is maintained by nerve impulses. If nerve impulses continue to activate the muscle, although voluntary effort has ceased, it is perhaps fair to assume that they arise either as the result of reflex activation or because of an imbalance of the mechanism of reciprocal innervation, whereby the central excitatory state of the motor center for the myotonic muscle might be thought of as incompletely abolished by the available inhibitory processes.

With regard to the latter view, we have found what appears to be definite negative evidence, whereas several facts brought out in our records lead us to favor the hypothesis that myotonic after-contraction is due to reflex activation. In a previous article[9] one of us demonstrated that a portion of the so-called "silent period" of the knee jerk in a normal subject is an inhibitory interval of relatively constant duration. In records obtained in the same manner for the rectus femoris muscle of these myotonic patients the inhibitory interval was of normal duration, but immediately after it a second knee jerk response often occurred. This suggests that in such a reflex contraction the inhibitory mechanism is functioning normally but that probably certain sensory end-organs in the muscle are hyperexcitable, so that their discharge outlasts the period of inhibition and results in the second knee jerk response. Such a condition of hyperirritability of sensory end-organs might well be responsible for the myotonic phenomena observed in such patients.

Figure 6 is presented to show that the electrical responses of single motor units, obtained by means of coaxial needle electrodes inserted into the flexor digitorum sublimis muscle of a myotonic patient, do not differ in any way from the

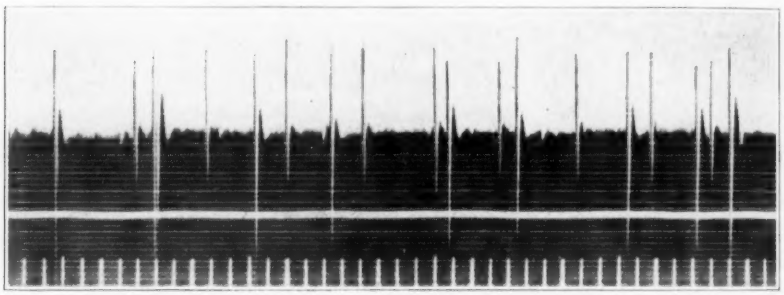

Fig. 6.—The action potentials from two single motor units in the flexor digitorum sublimis muscle of the patient with myotonia congenita during voluntary flexion of the middle finger against the myograph. The time unit equals 20 milliseconds.

regular, rhythmic responses of single motor units of a normal subject. This record, as well as all the others obtained from both patients, indicates that so far as the electrical responses are concerned, contraction is maintained in a normal manner. It is possible, as we have pointed out in connection with figures 3 and 4, that there is a tendency for a slower inception of contraction and an earlier onset of fatigue in the myotonic muscles, although this is certainly not marked.

As previously mentioned in the description of these cases, percussion of a myotonic muscle with a reflex hammer produces a myotonic reaction which forms a persistent groove or furrow along the lines of muscle cleavage. When coaxial needle electrodes are inserted into the muscle where such a groove is produced by percussion, the electrical responses of one or more motor units can be heard in a loud speaker as long as the contraction lasts. Figure 7 is a record of such activity and reveals several active units at the start. Emerging from this initial activity is the response of one unit more prominent than the rest, which seems to

9. Lindsley, D. B.: Am. J. Physiol. **109**:181, 1934.

be discharging about fifteen times per second but gradually slows down to three per second about four seconds later and finally reaches a frequency of about two per second (part *B*) just before ceasing to discharge, eight seconds after the initial stimulating tap with the reflex hammer. This is illustrative of the duration of myotonic activity following mechanical stimulation and shows the gradual slowing of the response of a single motor unit to a frequency (two per second) lower than has been demonstrated [10] in voluntary activity in several normal subjects.

The Effect of Drugs on Myotonia.—One of the difficulties in determining the effects of drugs on myotonic conditions in patients has been the lack of a precise objective measure of the duration of the after-contraction. Following the method used in obtaining the records of figure 3, we found that the electrical activity accompanying a myotonic reaction subsequent to a very strong hand grasp is relatively constant for each patient at any one experimental period. For instance, the duration of the electrical activity in the patient with myotonia congenita following the release of a strong hand grasp was consistently three seconds, whereas

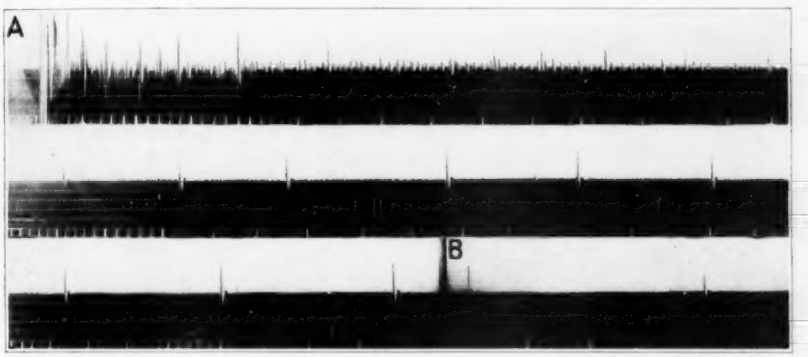

Fig. 7.—The action potentials obtained from a myotonic groove produced in the pectoralis major muscle of the patient with dystrophia myotonica. Several units are active at the start; one that is more prominent than the rest continues to discharge for eight seconds, gradually slowing. Three seconds elapse between the end of *A* and the beginning of *B*. The time unit equals 23 milliseconds.

that for the patient with dystrophia myotonica was approximately nineteen seconds. We found also that the amplitude and duration of the large groups of responses in the records of any one patient were relatively constant. Such a means of measuring the myotonic activity has therefore been useful in observing the effects of various drugs on the myotonic reaction of each patient. Control records were obtained at the start before the injection of a drug and others at regular intervals up to an hour after the injection.

In both patients calcium gluconate (1 Gm. in 10 cc. of water given intravenously) produced a significant decrease in the duration of the electrical activity. Likewise, the size and duration of the individual groups of responses immediately following the signal to release were diminished. In case 1 (myotonia congenita) a significant reduction in the total duration of the electrical activity accompanying

10. Lindsley, D. B.: Am. J. Physiol. **113**:88, 1935; **114**:90, 1935.

the after-contraction was apparent five minutes after the injection, but the greatest reduction (60 per cent) occurred forty-five minutes after the injection. The next record, taken sixty minutes after the injection, showed the total duration of the after-contraction to be lengthening again. At a later date an injection of calcium chloride (3 Gm. in 30 cc. of water) was tried, with similar partial reductions of the electrical activity accompanying the after-contraction. The severity of the myotonic activity was certainly reduced significantly, both subjectively and objectively. The most convincing effect was the reduction, by at least half, of the duration of the after-contraction produced by mechanical stimulation with a reflex hammer. Three observers and the patient agreed that the dissipation of the groove or the contraction produced in this manner was much quicker following the injection of calcium chloride than before.

In case 2 (dystrophia myotonica) a significant decrease in the total duration of the myotonic reaction was apparent nine minutes after the injection. In the same patient a record of the myotonic reaction produced by mechanical percussion of the pectoralis major muscle was obtained by means of coaxial needle electrodes inserted into the muscle. Figure 8 *A* is typical of several control records taken before the injection of calcium gluconate and shows a number of units which are

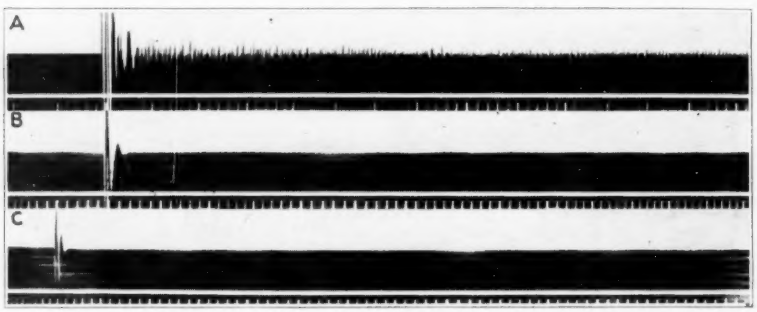

Fig. 8.—Same as figure 7. *A*, before the injection of calcium gluconate, showing many units active. *B*, two minutes after the injection, only one or two units being active (not visible on reduced print). *C*, five minutes after the injection, no electrical activity or myotonic reaction following the stimulating tap. The time unit equals 23 milliseconds.

persistently active throughout the record, following the quick tap on the muscle with a reflex hammer. Figure 8 *B*, taken two minutes after the injection, is characteristic of three records taken at that time and shows but a single unit active (very small on the original record and practically invisible in the reduced print). This unit was discharging at a frequency of twenty-two per second immediately after the tap but gradually slowed down to thirteen per second at the end of the record. Figure 8 *C*, taken five minutes after the injection, shows no electrical activity or myotonic reaction following the mechanical stimulation.

Among the other drugs that have been tried without manifesting any diminution of the myotonic reaction in the muscles of the forearm after strong clenching of the fist are ergotamine tartrate (gynergen), pilocarpine and epinephrine. In the patient with myotonia congenita ergotamine tartrate (0.25 mg. intravenously) produced a slight increase in the size and duration of the groups of impulses recorded immediately following the signal to relax and had no effect on the total

duration of the after-contraction. Pilocarpine hydrochloride (3 mg. intravenously) produced an increase in the size and duration of the groups of responses following the release signal and also increased the total duration of the after-contraction. Epinephrine hydrochloride (0.33 cc. of a 1:1,000 solution intravenously) in the patient with myotonia congenita caused a marked increase in the duration and size of the groups of impulses after the signal to release as well as in the total duration of the electrical activity. In the patient with dystrophia myotonica no change was produced by the epinephrine.

The patient with dystrophia myotonica after taking amino-acetic acid (30 Gm. per day) for two months reported only slight improvement in strength but no change in the active myotonia. Records for the muscles of the forearm after treatment with amino-acetic acid showed no diminution of the duration of the after-contraction.

COMMENT

Most of the theories advanced to account for myotonic phenomena in myotonia congenita or dystrophia myotonica can be classified as having a neurogenic or myogenic basis, although Thomsen, who first described myotonia congenita, stressed the psychic factors involved. Rosett [4] has also called attention to the incidence of neurotic and psychotic tendencies as associated hereditary factors which may be closely related to the muscular disorder. Jelliffe and Ziegler [6] in a recent publication have expressed a desire for a more thorough investigation of this aspect of the disorder.

The presence or absence of action currents during the myotonic reaction has been an important point in the argument of several authors either for or against a neurogenic or myogenic theory. Gregor and Schilder,[11] as early as 1913, thought that they were able to demonstrate by means of a string galvanometer that action currents were present during the myotonic after-contraction in patients with dystrophia myotonica. Later, Bürger and Schellong [12] also presented evidence that action currents persist during the prolonged phase of relaxation in myotonic muscles. Adie and Greenfield [5] and more recently Condorelli [13] have reported the absence of action currents during the after-contraction of active myotonia and mechanical myotonia and have agreed with Schaeffer,[14] Grund [15] and others that myotonia has a myogenic basis. A more recent study by Delmas-Marsalet and Bargues [16] has added little definite information one way or the other, since their records are not convincing, although apparently they concluded that both active and mechanical myotonia are accompanied by action currents.

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11. Gregor, A., and Schilder, P.: Ztschr. f. Neurol. u. Psychiat. **17**:206, 1913.
12. Bürger, M., and Schellong, F.: Ztschr. f. d. ges. exper. Med. **31**:82, 1923.
13. Condorelli, L.: Clin. med. ital. **59**:225, 1928.
14. Schaeffer, H.: Deutsche Ztschr. f. Nervenh. **67**:225, 1920.
15. Grund, G.: Deutsche Ztschr. f. Nervenh. **42**:110, 1911.
16. Delmas-Marsalet, P., and Bargues, R.: J. de méd. de Bordeaux **111**:235, 1934.

Contrary to the reports of Adie and Greenfield [5] and Condorelli,[13] whose results have led them to favor a myogenic theory of myotonia, we have been able to demonstrate the presence of action currents during the after-contraction of both active and mechanical myotonia. According to Adrian,[17] "there is general agreement that the action current denotes the passage of an impulse into the muscle fiber"; hence the presence of action currents in the muscle during the period of the after-contraction indicates that the muscle is receiving nerve impulses. Our results therefore militate against the theories that the prolonged relaxation has a purely myogenic basis, for were an increased viscosity of the muscle substance or some other abnormality of the contractile mechanism at fault, action currents should not appear throughout the period of after-contraction following the cessation of voluntary effort. Moreover, the fact that the muscle makes a normal initial relaxation before it "catches" (figs. 4 and 5) and then is released further by steplike gradations also precludes the concept of abnormal viscosity. If a condition of abnormal viscosity existed, the early phase of relaxation would hardly bear so close a resemblance to the quick, smooth descent of the normal myograph curve (fig. 4).

Condorelli [13] favored a myogenic theory, based on the hypothesis that the sarcoplasm itself produces "tonus" as a type of contraction distinct and separate from the clonic variety and is characterized by the absence of action currents. This view, so far as we are aware, is without experimental evidence to support it and is not generally accepted (compare Fulton,[18] Cobb [19] and Cobb and Wolff [20]).

The fact that following the cessation of voluntary effort the muscle remains contracted for several seconds and continues to receive impulses might be interpreted as the result of an imbalance of the mechanism of reciprocal innervation whereby inhibitory processes are insufficient to abolish residual excitatory ones. As a consequence, a central excitatory state persisting above the threshold for certain motor units of the "motoneuron pool" might result in a continued discharge of those neurons, thus maintaining some tension in the muscle in the absence of voluntary effort.

Several points brought out in this paper do not seem to be in accord with this view. In the first place, the duration of the after-contraction, which was three seconds in one case and nineteen in the other, seems to be much longer than one would expect for the dissipation of an excitatory

17. Adrian, E. D.: Lancet **1**:1229, 1925.

18. Fulton, J. F.: Muscular Contraction and the Reflex Control of Movement, Baltimore, Williams & Wilkins Company, 1926.

19. Cobb, S.: Physiol. Rev. **5**:518, 1925.

20. Cobb, S., and Wolff, H. G.: Muscle Tonus, Arch. Neurol. & Psychiat. **28**:661 (Sept.) 1932.

state unless it were continually being replenished by centripetal impulses, as we are inclined to believe is the case. Furthermore, the action currents which appear in groups or volleys, as shown in figure 3, and also those of figure 5, which accompany the steplike relaxation curve, are certainly suggestive of motor discharges of reflex origin rather than a smooth continuous discharge of motor neurons, such as one might expect in the dissipation of an excitatory state. As already mentioned, the inhibitory interval following the knee jerk in these patients was of normal duration and leads us to believe that there is no defect in the inhibitory mechanism. Clinically the knee jerk response in both cases was normal and not particularly hyperactive, but our records often reveal the presence of a second knee jerk response, which indicates an unusual "rebound" effect, probably due to the prolonged after-discharge of certain hyperexcitable sensory end-organs in the muscle, the central effect of which is excitatory and outlasts the period of inhibition.

Matthews [21] and Talaat [22] have shown that very slight changes in the ionic concentration of a fluid medium, such as a deficiency of calcium in Ringer's solution in which a preparation is bathed, will cause an increase in the excitability of the sensory end-organs and will prolong the discharge of nerve impulses. Under such conditions a single stimulus may cause a persistent discharge of the sensory end-organs. If it is assumed that the sensory end-organs in the muscles of myotonic patients are hyperexcitable owing to a chemical condition of the body fluids, it is reasonable to suppose that reflex contractions of a muscle could be set up by the normal changes of pressure and the stretching which must occur when some fibers have begun to lengthen and others have not as relaxation begins. It is also possible that the muscle spindle, which has been shown to have both sensory and motor innervation, will in an overexcitable state give persistent sensory after-discharges in response to the motor impulses it receives. In such an event one might reasonably expect the sensory discharge to result in reflex activation of the muscle after the voluntary effort has ceased. In fact, it is probably the sensory response of the muscle spindle to its own motor discharge which accounts for the reflex myotonic effects rather than passive stretch.

Regardless of how the sensory discharge is set up in hyperexcitable sensory end-organs, the delayed relaxation is apparently due to the reflex activation of the muscle and will subside only when the sensory end-organs become fatigued or have reached a state of adaptation so that the centripetal flow of impulses is diminished. The fact that "warming up" a muscle by preliminary activity reduces the duration of the after-contraction, as was shown in figure 3*C*, lends support to this hypothesis.

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21. Matthews, B. H. C.: J. Physiol. **71**:64, 1931.

22. Talaat, M.: J. Physiol. **79**:500, 1933.

In practically all the records, such as those of figure 5, some single motor units are active as the myograph curve makes its first downward swing. These units are reflexly active, we believe, as the result of after-discharges in the hyperexcitable muscle spindles which have been receiving, along with other muscle fibers, the motor discharges of the original voluntary effort. As voluntary effort ceases, a persistent sensory after-discharge from these muscle spindles continues. The residuum of contraction maintained by such reflex activation results in the plateau-like levels which occur as the muscle relaxes. With the "catching" which occurs at each of these levels of relaxation, stretching and other forms of stimulation result in further reflex contractions. Thus, duration of the myotonic contraction depends on the irritability of the sensory end-organs and the degree of stimulation, the latter depending in turn on the strength of the contraction and the stretch and pressure influences which accompany relaxation in hypertrophic muscles.

As mentioned heretofore, determinations of the calcium content of the blood in both of our patients showed no deviation from the normal range to which could be attributed the hyperexcitability of the sensory end-organs in the muscle. We believe, however, that it is entirely possible that other chemical constituents of the blood, either in excess or in deficiency, might be responsible for the condition of hyperexcitability. It was thought that even though the calcium content was within the normal range, increasing it still more might decrease the excitability of the end-organs. Intravenous injections of calcium gluconate seem to have this effect. Although the effect of injections of calcium lasted only about an hour and only partially diminished the after-contraction, the effect was thought to be significant so far as our hypothesis is concerned. Attempts to give calcium by mouth or to increase the calcium content of the blood by means of injections of parathyroid extract [23] have not succeeded in raising the calcium content of the blood sufficiently to have the desired effect.

It is of interest in this connection that Pamboukis [24] reported temporarily relieving two myotonic patients and permanently relieving another by the administration of calcium chloride and acetylsalicylic acid. In another case he reported beneficial results after forty injections of solution of posterior pituitary. He also reported, in agreement with our findings, that pilocarpine increased the myotonia, although Monrad-Krohn [25] has reported a case of myotonia in which a diminution of myotonic symptoms was noted following the injection of pilocarpine.

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23. The preparation used was parathormone, the product now called parathyroid extract-Lilly.

24. Pamboukis, G.: Schweiz. Arch. f. Neurol. u. Psychiat. **26**:84, 1930.

25. Monrad-Krohn, G. H.: Acta psychiat. et neurol. **5**:241, 1930.

It is our belief that myotonia is not a disturbance of the sarcoplasm but a normal contraction of muscle initiated and maintained by nerve impulses. The fact that the contraction persists after the cessation of voluntary effort or of mechanical or electrical stimulation, we think, can be accounted for on a reflex basis and probably results from stimulation of hyperexcitable sensory end-organs in the muscle, which may continue to discharge even after the stimulus has been removed.

SUMMARY

A case of myotonia congenita and a case of dystrophia myotonica are described. The myotonia, which appeared to be the same in the two cases, was studied electromyographically.

The after-contraction of myotonic muscles, which persists after the cessation of voluntary effort or brief mechanical stimulation, is accompanied by action currents. This indicates that the phenomenon is neurogenic rather than myogenic.

The evidence presented favors the hypothesis that the after-contraction of myotonia is of reflex origin and is due to the persistent discharge of hyperexcitable sensory end-organs in the muscle.

Injections of ergotamine tartrate, pilocarpine hydrochloride and epinephrine hydrochloride have proved ineffective in relieving the myotonic condition, although calcium gluconate and calcium chloride have significantly reduced the amount and duration of the after-contraction.

DIFFERENTIAL DIAGNOSTIC TYPES OF SUICIDE

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Prevailing views on suicide have been transmitted with comparatively little change from generation to generation for the past one hundred and fifty years. Viewed with awe and a certain psychologic embarassment, suicide appears to the present-day psychiatrist as it did in the past, as an abnormality of mental reactions, or more specifically as an aberration of the instinct of self-preservation, arising from what it has become customary to call undue depressive affects. This point of view, while couched in present-day terms, differs little from that expressed by several generations of medical psychologists, who in turn inherited it from the tradition originated by Saint Augustine.

Saint Augustine was one of the first, if not the first, to postulate that suicide is a sin![1] And sin in those days was a voluntary surrender of one's reason to the pressure of evil, i. e., a sort of intellectual aberration of one's will. The Council of 452 A. D. decreed suicide to be the work of the devil, and as it is well known, many works of the devil have come down to the present in the guise of severe neuroses and psychoses. Hence, the intellectual and moral evaluation of suicide has changed comparatively little, and this estimation, humanized and couched in the scientific terms of modern psychopathology, remains negative; to put it in the words of one of the most recent students of suicide: "It is opposed to the instinct of self-preservation and is therefore a typical perversion."[2] The so-called common sense psychology, while always admitting that suicide is an act of unusual psychologic meaning, tries to explain it by the supposition that it must be an act of despair and possibly one of great courage. However, one hears it as frequently estimated as an act of cowardice. In other words, popular opinion is ambivalent on the subject and views it with a mixture of contempt and admiration. This fact is of more than passing scientific value. One might recall in this connection that on the statute-books of more than one country even today suicide is considered a crime and that Plato almost twenty-five centuries ago was inclined to believe that suicide is a dishonorable act, for a citizen has no right to deprive society of his civic life without the permission of a magistrate. This tradition of disapproval trans-

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Read at the meeting of the New York Neurological Society, April 3, 1934.

1. Augustinus, D. Aurelius: De civitate Dei, I, XVI.

2. Achille-Delmas: Psychologie pathologique du suicide, Paris, Félix Alcan, 1932.

formed itself in modern times into recognition of an obscure illness and a tendency to limit the suicidal reaction to a group of "insane" persons. That is probably why there has been a constant search for a special pathologic basis for suicide, which some persons see in a singular disturbance of the affective field, others in constitutional predisposition (since many suicides occur in the same family) and still others in specific, although as yet undifferentiated, changes in the brain (in cases of suicide in the involution period of a depression and in those occurring occasionally among persons with arteriosclerotic psychoses and less frequently after severe organic trauma [3]). One therefore frequently hears the inquiry whether it is at all possible for a person to commit suicide without the development of a severe pathologic depression, and one wonders whether the unexpected suicide of some one who appeared normal until the last minute is not, after all, the result of a sudden psychopathologic aberration, which might be called pathologic depression, despair, sense of hopelessness, etc. Be that as it may, it is clear that the problem of suicide from the scientific point of view remains unsolved. Neither common sense nor clinical psychopathology has found a causal or even a strict empirical solution. Most recent statistical studies of suicide have shocked the common sense school by demonstrating that there are more suicides in the community in days of prosperity than in periods of economic depression,[4] and clinical psychiatrists look askance at the fact that, while it is true that patients with depressive psychoses more often commit suicide than other mentally sick persons, a number of suicides occur when the depressed person appears to be convalescing and all but recovered from his depressed state. Moreover, a number of patients who neither are classified as persons with depressions nor behave as such commit suicide at a time when even the experienced clinician had little reason to suspect its possibility. It is hardly necessary to state that neither preventive measures nor scientific causal therapy is at all possible under these circumstances and that only a comparatively small group of patients with severe depressive psychoses can be recognized as potentially suicidal and placed in the protective environment of a hospital.

It is my purpose in this communication to raise and to answer only partially rather than to solve the following questions which suggest themselves: What kind of persons are likely to commit suicide? What is it, i. e., what are the psychologic processes that lead them to suicide? Are there any objective data, signs or manifestations which would lead one to suspect the possibility of suicide before the act is committed?

3. Jankau, V.: Selbstmord nach Unfall, Ztschr. f. d. ges. Neurol. u. Psychiat. **130**:148, 1930.

4. Dublin, L. I., and Bunzel, Bessie: To Be or Not to Be: A Study of Suicide, New York, Harrison Smith & Robert Haas, 1933, p. 102.

What inferential material has the clinician at hand that might enable him to think in an empirical way of precautionary, if not preventive, measures?

The past twenty-five years have witnessed the development of a deeper insight into the purely psychologic processes of the normal and the abnormal person, and one has learned to view with respect the instinctual drives of man as they present themselves to him, particularly in their unconscious constellations. In other words, a man's development and his instinctual and emotional reactions to the various stages of his development are now recognized as powerful dynamic factors in his life. While the etiology and nature of many of these for the most part unconscious reactions are still obscure, a mass of empirical data has been accumulated which permits one to observe some unconscious processes and to evaluate their respective emotional and dynamic importance in a given case. It would appear logical, therefore, to inquire what the newer psychopathology, which is based on psychoanalysis, has contributed or has to contribute to the problem of suicide.

It is of interest in this connection to quote the words with which Freud summarized the result of a symposium on suicide held in Vienna in 1918:

> Despite the valuable material obtained in this discussion, we have not succeeded in arriving at any definite conclusion. We should like to know above all how it is possible that one overcomes such an extraordinary powerful instinct as the life instinct; is this possible only as a result of disappointment to which the libidinous drives are subjected, or because the ego, pursuing some interest of its own, renounces any self-assertion in life? Perhaps our failure to find an answer to this psychological question is due to the fact that we have no way of obtaining it; by this I mean that in this problem we are able to utilize as a point of departure only the comparison of the clinically known state of depression with the affect accompanying mourning. However, we are totally ignorant of the affective processes of depressions and of the fate of the libido in this illness; nor is the affect of the mourner yet understood analytically. Let us therefore refrain from forming an opinion until the time comes when experience will have solved the problem.

In other words, the psychoanalytic symposium on suicide at Vienna failed to formulate an adequate answer to the vexing question of what the psychopathology, or rather the psychology, of suicide is. The consensus at that time was that every suicidal person is the victim of strong aggressive impulses which he fails to express outwardly and which he, as a result, turns inward, i. e., on his own self. However, this rather skilful formula, which has since become popular in clinical psychiatric circles, failed to establish a definite clinical or even a theoretical criterion which would throw light on the problem of suicide. Fundamentally the formula was correct, but it was too general to be of any real value,

for while it is true that any one who wants to or who does commit suicide suffers from strong aggressive (unconscious) murderous impulses, these impulses are so universal that according to this formula alone the whole world should be in danger of committing suicide, which is obviously not true (Federn).

This unsatisfactory state of understanding was partially ameliorated by the further studies of Abraham, who investigated a number of depressive psychoses, and by Freud, who shortly after the symposium on suicide subjected to careful scrutiny the mental processes of persons with depressions as compared with those of mourners. As a result, a wealth of clinical data was collected, and a better understanding of the depressive psychoses was gained. This understanding can be briefly summarized as follows:

The person suffering from a pathologic depression has a specific set of unconscious fantasies which determine his mood, his whole illness, and a characteristic emotional attitude toward the world which determines his behavior. He identifies himself with another person whom he once loved and then hated; he then loves and hates himself and falls victim to this internal raging battle. The identification, however, in order to produce a depression, is of a special nature, and is based on a particular type of fantasy, a special mechanism. This can be described as follows: The subject is under the dominant influence of a fantasy that he swallowed the once loved and then hated person; he becomes that person and hurls the whole mass of his hostility on this internalized person; the process of being hostile to the internalized person or persons is perceived as depression, self-depreciation and self-hatred, while the act of murder of that person or persons is the act of suicide. As one can readily see, this set of formulations based on the study of the fantasy-life in manic-depressive psychoses is merely an empirical statement of what was observed; it does not, of course, explain suicide. It is of interest to note, by way of illustration, how the aforementioned mechanism of depression can be clinically demonstrated: Thus, it was found that the self-accusations of some depressed persons are frequently literal reproductions of accusations which at one time had actually been directed against the person whom the depressed patient internalized psychologically. Abraham cited the case of a girl who accused herself of being a thief in the same terms which had once been used against her father when, during her young girlhood, he was suspected of having stolen. Moreover, the subject's unusual sadism is taken over by his own conscience, which relentlessly drives him into deeper and deeper depression.

Freud's point of departure was a comparative study of the affects accompanying mourning and depression. His conclusions can be summarized as follows:

In grief which follows an actual loss of a love object, the ego suddenly finds itself in possession of a great amount of free psychic energy which heretofore was bound to the lost object. This free energy cannot naturally reattach itself at once to a new object, for the memories of the lost person keep the subject in a state of tension which cannot at once be overcome. The host of incidents and details related to the lost person, which had been previously perceived as pleasant, become a source of psychic pain, because the object to which love was attached is gone and the psychic energy has to be freed from what was real and has become unreal. The subject at no time loses his capacity for object relationship, but the loss of the object and the psychic energy so painfully freed (made unattached) cause him to withdraw (temporarily) his interest in life. Only after the painful process of weaning from the lost object is completed does the process of reattachment begin and the mourner find his interests in life gradually revived and reestablished. At no time is the sense of loss absent from consciousness.

The inner, affective picture of a pathologic depression is different. Seldom, if ever, does one find a sense of actual loss. To be sure, the death of a loved person may be a precipitating cause of a depressive psychosis, but throughout the depression the complaints of the patient center around other things: He feels dull; he is self-deprecatory and self-accusatory; he has no memory, no mind, no body, etc. The whole trend could be characterized as a complaint that the ego is greatly impoverished; it has lost its tone of life. What happened? Normally, one's ego is turned toward the outside world, and one's psychic energy is bound to it; in a depressive psychosis this energy is withdrawn from reality; this energy, this love for reality, not only becomes free from attachment but renounces any object relationship; it turns toward itself as an object. In other words, it makes its own ego the object; it becomes self-centered, self-loved, self-contained and self-indulgent; that is, it regresses to the earlier level of development known as the narcissistic level. The object or objects which once were ostensibly loved the ego takes into itself, so to speak; it identifies itself with them by way of the mechanism already briefly described and technically known as oral incorporation. The whole process will remain obscure and misunderstood, however, if one does not bear in mind the following fundamental characteristic which is pathognomonic of pathologic depression: The object, the person from whom the depressed person has withdrawn his love, has never been fully loved; unconsciously, and at times half consciously, there always existed a strong undercurrent of dislike, hatred and aggressiveness toward that object. It is this ambivalence, with the negative attitude predominating, which is characteristic of pathologic depression. Consciously the patient may continue to profess love for the object or to project his own aggressiveness, which then appears in

the form of anxiety. All psychiatrists have known patients who without apparent rational grounds begin to fear that the husband, wife, child, mother or father might die, might fall a victim to a terrible illness, etc. However, the brunt of the unconscious sadistic hatred and aggressiveness is borne by the ego itself, since it has identified itself with the object and since, in addition, the subject's conscience produces a severe sense of guilt because of the criminal wishes. The result of this structural affective arrangement is a psychic conflict presenting the typical picture of a pathologic depression.

To recapitulate briefly: In a case of a pathologic depression the patient, through identification with a person toward whom his feelings have always been highly ambivalent, loves and hates himself. Since his own ego has become his love object, he feels detached from reality and therefore experiences a sense of poverty of the ego. The unconscious sadism originally directed against the object, reenforced by a sense of guilt, produces the singular phenomenon of the person's becoming sadistic toward himself. Freud [5] stated:

> It is this sadism, and only this, that solves the riddle of the tendency to suicide that makes depressions so interesting and so dangerous. As the primal condition from which the life instinct proceeds, we have come to recognize a self love of the ego which is so immense, and in the fear that rises up at the threat of death, we see liberated a volume of narcissistic libido which is so vast, that we cannot conceive how this ego can connive at its own destruction. It is true we have long known that no neurotic harbours thoughts of suicide which are not murderous impulses against others redirected upon himself, but we have never been able to explain what interplay of forces could carry such a purpose to execution. Now the analysis of depressive psychoses shows that the ego can kill itself only when the psychic energies, which were originally attached to the object, have been withdrawn upon the ego and the ego can treat itself as an object, when it is able to launch against itself the animosity relating to an object—that primordial reaction on the part of the ego to all objects in the outer world. Thus in the regression to narcissistic object choice the object is indeed abolished, but in spite of all it proves itself stronger than the ego's self.

It is of historical interest to note that the concept of hostility first turned outward against the world and then inward against oneself was the basic idea in the formulations on suicide by the student of a century ago, who sensed it intuitively without being conversant with the mental mechanisms with which modern psychopathology deals. Thus, among others, Tissot in 1840, in his monograph, entitled "De la manie de suicide et de l'esprit de la révolte," postulated that "suicide and revolt are but the double expression of one and the same cause, a double symptom of the same moral malady." Evidently, at least a part of Freud's hypothesis finds its corroboration retrospectively in the sociologic studies of the past. However, despite the considerable insight gained in regard

5. Freud, S.: Collected Papers, New York, 1924-1925, vol. 4, p. 162.

to the mental processes in cases of suicidal depression, the trend observed in prefreudian psychopathology persisted in psychoanalysis also, viz., that suicide is the pathologic outcome of a pathologic state called depressive psychosis. This trend led Freud to state:

> It is remarkable that in contradistinction to depressions, the compulsive neurotic never makes a suicidal attempt; he gives the impression of being immune against the danger of suicide, more so than the hysteric. We understand, of course, that it is the preservation of the object that serves us as a guaranty of security of the ego.

A critical evaluation of all these views is possible only through the examination of clinical material. It is hardly necessary to cite many instances in corroboration of the fact that strong hostilities are operative in cases of depressive psychoses and that these hostilities are characteristically directed by the patients against themselves.

REPORT OF CASES

Case 1.—A woman, aged 28, who had always been unusually devoted to her father and whose mother died when the patient was 13, suffered for several years from gastro-intestinal discomfort, periods of irritation and chronic discontent. The only companionship she ever found satisfactory was that of her father. She made many attempts to "be like other girls," but was unable to make herself enjoy the attentions of men. She would take two or three drinks to stimulate herself "to greater courage," but she experienced only disgust when a young man made love to her. One day, a short time before her father was to come home from work, she "decided to end it all;" she put on her prettiest silk brocade kimono and her nicest mules with the conscious intention of looking as attractive as possible; she had a definite wish that her dead body should be pleasing to her father's eye. She turned on the illuminating gas. A short time later she was discovered unconscious by her father. She was taken to a hospital. On recovery from the after-effects of mild gas poisoning, she was transferred for treatment to a hospital for patients with mental disease. She was suffering from depressive neurosis of long standing.

Case 2.—A woman in the late thirties, mother of three children, had been in low spirits for some time. In a period of a few months she became increasingly tense, agitated and restless. She complained of insomnia and peculiar sensations in the wrists. Finally, she decided to commit suicide. She slashed her wrists and lost a great deal of blood, but she was saved from death and brought to the hospital. The wounds healed soon, but the emotional state continued with great severity. She wept, groaned, paced the floor and slept little. She professed to have great love for her children and especially for her husband. They were far away in their home in the West. One day, escaping for but a moment the vigilance of the nurse and with a swift, impulsive motion barricading the door by means of her dresser, she broke the window-pane and cut her throat with the glass. As a result of immediate medical attention she failed to die this time also. A few months passed; the patient, while still tense and agitated at times, became more sociable and interested in things outside herself and began to read; she smiled frequently. One day she concealed a piece of glass and in a cheerful, simple manner asked the nurse to bring her a glass of water. While the nurse

went out to comply with the request she cut a deep wound in the right side of her neck. A few minutes later she was found cyanotic, almost exsanguinated and gasping. As soon as medical intervention revived her, she began to struggle with surprising violence, demanding that she be allowed to die. However, she recovered, and she was later studied in detail until the drive to suicide receded and then apparently disappeared.

CASE 3.—A woman, aged 48, had been nervous, tense and dissatisfied for many years. She had always been afraid of pregnancy and was sexually frigid; as a result her marital life was unhappy. She acquired the neurotic habit of consulting many physicians, whom she called on with increasing frequency. She had examinations every time that she suspected she was pregnant. These suspicions and apprehensions she experienced before, during and after the menstrual periods. Although she was a Roman Catholic, she insisted on the use of contraceptives. Yet she remained dissatisfied. She insisted on having curettage done when none was indicated. She insisted that a surgeon tie the uterine tubes. When this was done she began to worry about possible infection. At one time she thought she had syphilis. She had a record of several residences in general hospitals and in institutions for patients with mental diseases. At the age of about 46 she reached the point of general hypochondriasis; she had, she claimed, no movement of the bowels; her intestines were "dried up." She frequently carried a piece of a fragile cake of sand, claiming that this was a piece of her "dried-up intestine"; her brain too was "dried up." She had her own theory of why she looked well physically and gained in weight despite her alleged afflictions. She was agitated and cried. About two years elapsed without bringing appreciable change in her general condition and demeanor. She began to complain in a demanding, aggressive manner that no one was interested in her and that she was abandoned or neglected by every one. On one or two occasions she stated that she was going to die soon. At no time did she express any direct suicidal thoughts—but one evening she suddenly hanged herself.

CASE 4.—A physician, aged 36, had a particularly great fear of increasing the size of his family and thus the burden of his financial obligations. Being a Catholic, he was equally fearful of the use of contraceptives, and the fecundity of his wife served constantly to deepen and sharpen his conflict. One day, one of his little sons died of an acute illness. He was sad, but his unconscious attitude was betrayed when one day the following exclamation inadvertently slipped from his tongue: "One mouth less to feed!" From that time he fell into a typical suicidal depression. He hanged himself and was revived with difficulty. After he was brought to the hospital, he persistently tried to escape the vigilance of the physicians and nurses in order to kill himself. Long therapeutic talks with his physician revealed an extremely strong unconscious wish that his children should die, a deep sense of guilt because of it, hostility and a murderous attitude toward others whom he thought he loved and a strong ambivalent, incestuous attitude toward his sister. Not only did this patient turn the hostility on himself, actually loving no one, but his deep sense of guilt supplied additional energy for the self-destructive impulses.

Comment.—One may postulate that every case of potential suicide must bear clinical evidence of strong unconscious hostility combined with an unusual incapacity for love of others, and if this evidence is found in a case in which depressive features appear suicide becomes

a real danger, even though the patient may never express the wish for self-destruction.

The girl in case 1, who attempted to die by gas poisoning, was studied by means of direct psychoanalytic approach. It was as surprising as it was illuminating to find how poor her capacity for love had always been. She was a chronic masturbator; she was unable to (unconsciously she would not) fall in love. She always played the rôle of a boy and had wished to be one ever since she could remember. She hated her mother. Her father, whom she professedly loved to the exclusion of any one and anything else, she also hated unconsciously. She never forgave him her disappointment when he first preferred her mother and then paid attention to other women after the mother died. She loved him, it appears, merely because he loved her, because "his love was safe," as she put it, i. e., because his love served her as a pretext for not loving others and as a protection against her seeking love elsewhere. Her brother, too, had always been to her the object of envy and hatred because he was a boy and she was not. So far as she played a masculine rôle in life (hence showing unconscious homosexual leanings), she identified herself with a hated brother and with a father to whom her attitude was extremely ambivalent. She withdrew her love from others and turned her great hostility against herself. Since she had a deep-seated sense of guilt and also hated her mother, with whom she partially identified herself and who was dead, it is clear that there were in this case more than the usual prerequisites for a strong suicidal trend.

The woman in case 2, who attempted suicide three times, was also studied in detail. Her peculiar history also led to the development of a number of extremely exaggerated hostile impulses, which were directed partly against her children but primarily against her husband. Her marriage was not happy. She was the boss, the provider and the brains of the family. She married the man from unconscious spite, although he was inferior to her socially and intellectually. She never loved him. She had always been a tomboy—a mastering, independent, aggressive person. The more intimate history of her childhood and her psychologic development from early childhood through adolescence revealed an increasingly growing hostility toward men (her father and younger brother at first), and her depression and suicidal drive were only an expression of that unbounded sadism which was directed against herself—an ego which had little love to spare for others.

Case 3 is a typical example of the same psychologic orientation. The patient loved no one. She was frigid. She wanted no children. Toward her husband her feelings were strikingly ambivalent. Her sadism, which was turned on herself, found for many years a partial outlet in delusions of being infected and in her frequent self-imposed surgical operations. In a hospital for patients with mental

disease her hostility toward the world and other persons found much less outlet than in the outside world. Her demands for surgical treatment remained unheeded; her hatred found expression only in the projection, "No one cares for me." Her incapacity to attach any of her psychic energies to things outside herself was so striking that, while she was not deeply depressed or retarded and was rather active, she was unable to interest herself in any occupational work, a nurse or another patient. She became a separate world in herself within which blind animosity was raging, and she hanged herself.

It is hardly an exaggeration to state that the clinical knowledge of suicide is exhausted at this point, because tradition has emphasized that depression means potential suicide and if a person with apparent absence of depression commits suicide, one believes that one must have lacked in diagnostic acumen and overlooked the "depressive features" in the case. This self-reproach is justified only so far as one has overlooked the suicidal potentialities but not in the questionable diagnosis of depression. The woman in case 1 at no time presented an appearance of classic depression; the condition fell more conveniently under the rubric of neurosis and was nearer the type of obsessional neurosis than any of the other recognized diagnostic appellations. The patient was never retarded, and at no time did she betray the ruminative type of self-accusations. Also, the majority of her symptoms were of a physical nature, consisting of such complaints as "indigestion," constipation, exaggerated menstrual pains and severe abdominal discomfort, which at one time (when her father took another woman home after a social function) became so severe that an operation for appendicitis was performed as an emergency and the appendix was discovered to be normal. Evidently this case was not an instance of a depression, i. e., not one of a depressive psychosis, despite the fact that the patient was suicidal. I shall postpone for a moment the search for diagnostic and prognostic features in this case to bear in mind that in the absence of a typical depression there is no guaranty against possible suicide. In other words, patients with other than typical depressions are likely to commit suicide, and in the light of present-day clinical experience Freud would not subscribe to his original statement that neurotic patients, particularly those with obsessional (compulsive) neuroses, appear to be immune to suicidal impulses.

The same conclusion is reached from a review of case 3. The patient, like the girl in case 1, was not depressed and showed throughout the greater part of her illness an unusually aggressive instead of a depressive mood; she was sarcastic and unpleasant. Both patients gave the impression of having little if any trouble in giving vent to their hostility and rancor, so that one wondered at times whether they had enough hostility left "to turn on themselves" and commit suicide. I shall again reserve

judgment until later as to what psychologic dynamic factors were operative in these cases in the direction of self-destruction and shall emphasize that the woman in case 3 had a misophobic, self-indulgent compulsion neurosis of long standing and not a depression.

To add to the illustrative series of suicides by persons who did not suffer from depressive psychoses, case 5 may be cited.

Case 5.—A man, aged 52, who was a rather successful and moderately affluent lawyer, was married and had one daughter to whom he was greatly attached when she was a child. His wife was neurotic and frigid. He was a somewhat worrisome, self-centered man, who spent little love on others. He apparently disliked his wife and had unconscious wishes to get away from her, to part from her. He expressed this trend by means of a projection. He became convinced that she was untrue to him with another man, his friend. This was never proved; moreover, there was no presumptive or inferential evidence of the wife's unfaithfulness, except his own assumptive conviction. Acting on this conviction, he established a relationship with another woman, which lasted for years until he came to a hospital for patients with mental disease. Yet his sexual demands were not great; his really gratifying mode of sexual expression appears always to have been masturbation—a habit he had never abandoned since childhood. He started to worry about his health, his heart, his head, the possibility of cancer, etc. His wife (with or without the insistence of the patient but at any rate with his assent) adopted a little girl. The patient liked the child, fondled her and loved her more than would seem normal. He later admitted rather obliquely to having a pedophilic trend. His aggressive impulses soon appeared through the devious path of projection—he became obsessed with the idea that the child might become infected. If a splinter got into her hand or if she stepped on a nail, tetanus might develop; if she was not careful on the fourth of July an explosion of fireworks might burn or infect her, since powder might contain tetanus bacilli. He wrote endless instructions on how to protect her; he changed them presently, for he had forgotten to add an important detail; he then telegraphed home added instructions. He was unable to make up his mind about anything; his ambivalent attitude invaded every field of his activity, so that at times he did not actually know what he wanted, since "yes" and "no," "right" and "wrong," "good" and "bad" appeared almost simultaneously in his conscious attitude toward things. He appeared dull, mildly depressed and preoccupied, even trembling; he stuttered at times, since "to say" and "not to say" were also simultaneous impulses with him.

One day he observed a small hole in the wire screen of a window on the second floor. (His room was on the ground floor.) He became interested in it and made it larger. Little by little, it became large enough for him to crawl out. He recalled that when he saw the hole for the first time a thought passed through his head: "Some one might some day tear it open and get out and injure himself." He crawled out, stood for a moment on the ledge and impulsively jumped off. A few minutes later he was heard calling to a passer-by for help. His voice was peculiarly full of cheerful vigor. While his face was covered with blood, his forehead wounded (the bone was unscathed), his legs wounded and one ankle dislocated, his eyes looked cheerful and almost happy, and he repeated with transparent pleasure: "Now I think I am going to be cured. I am going to get well now." Throughout the period of being bedridden, when he must have suffered a great deal of pain, the patient made no complaints and appeared singu-

larly comfortable. However, the obsessional and depressive symptoms reappeared gradually after complete recovery from the injuries he had sustained.

Comment.—This clinical picture leaves no doubt of its being that of a compulsion neurosis, and one may add that, suffering as this patient was from a severe sense of guilt as a result of unconscious hostility and forbidden sexual impulses, he appeared to obtain relief from the self-imposed punishment; this end achieved, he naturally became more cheerful and hopeful. To be sure, the unconscious trend for which he judged himself did not disappear, but it became in some way less unpermissible or more permissible, since he paid for it with his own blood. This psychologic mechanism is met frequently in patients who try unsuccessfully to kill themselves and in some transgressors of the law when they are punished by the State. The same mechanism came to light in the case of the suicidal physician in case 4. After a desperate attempt to commit suicide (by sticking a needle in his heart), he suddenly began to have nocturnal emissions, accompanied by incestuous dreams about his sister. He was greatly attracted to her before he married. Unconsciously, he never abandoned this attachment. His sense of guilt because of this was great, but once he paid for it by means of self-punishment, as a result of which he might have died, his conscience was sufficiently appeased to permit the sinful fantasy to enter his dream.

The sudden recurrence of the incestuous impulse in persons with strong suicidal drives is of paramount diagnostic importance. Hence, one can hardly overestimate the importance of a careful anamnestic study of all direct and indirect evidence and inferences of too great an attachment and too intimate a relationship, no matter how conventionally correct, of the patient to any member of the family which keeps him under the constant strain of having his incestuous wishes stimulated and inhibited at one and the same time. One may recall in this connection the woman in case 1 and her exclusive relationship to her father.

In the light of this clinical evidence that not only depressive psychoses but neuroses are liable to result in suicide, I may also state that schizophrenia does not represent an exception in this respect. Thus, I may mention a case of advanced catatonia of many years' duration.

CASE 6.—A man, aged 31, was considerably deteriorated socially, intellectually and emotionally. He often indulged for weeks in tearless catatonic whining or manneristic, ceaseless shouting. He defecated and urinated spontaneously like a child. He was fed by a tube for several years; he could not wear clothes except for occasional intervals of a day or two; he gave evidence of a scattered, fragmented, fantastic trend; he heard voices, called for mother incessantly and drooled constantly. With his fingers he loosened teeth and pulled them. Not infrequently he made other attempts to injure himself; he beat his head against the wall or the edge of the bed, cracking his scalp; he plunged from bed head downward. These attempts were vigorous and impulsive. At times he struggled with three or four husky male nurses to get free in order to kill himself. He shouted

that he wanted "to die, to go to God." The death impulse was so strong at times that it gave the impression of a wild, insatiable urge.

Comment.—While the condition in case 6 may appear too exaggerated to be of common occurrence, one is justified in stating that suicidal trends and acts in a patient with schizophrenia are not unusual. Suicides in these cases are at times among the most difficult to deal with and among the most puzzling. For instance, a patient with acute schizophrenia arrived in the hospital in a somnolent state, which gave the impression of the early stages of stupor; yet he at times aroused himself long enough to reveal a fairly well systematized paranoid trend: His associates were after him; they were all plotting against him; they wanted to deprive him of his job and standing; he thought of them as being mostly Irish Catholics who rose against him, a descendant of an old Protestant Southern family. He tried to castrate himself with a razor. He wished to kill himself, and for a time he presented one of the most difficult problems of management.

The persistence of the impulse to die and its violence are, however, by no means the outstanding clinical features in cases of suicidal schizophrenia. On the one hand, a number of depressions (such as that in the case of the woman who cut her wrists) show the same features, while on the other, the patient with schizophrenia is likely to serve little if any notice of his suicidal intention. For instance, an unmarried man in the early thirties, suffering from paranoid psychosis, displayed extreme jealousy in the acute stages of the psychosis and at the same time an intense flight from any real love for a woman. After several months in a hospital for patients with mental disease he appeared quieted, became apparently more serene and seemed to be convalescent. He showed no external ill effect from the visit of his fiancée one afternoon and continued with the routine of the hospital in his usual manner. He was seen that evening playing billiards with other patients. He greeted the physician with a free, apparently natural smile and retired a little later in good spirits. He was found early in the morning crouched and hanging on his necktie by the bedside.

Evidently, there is no single clinical entity recognized in psychiatry that is immune to the suicidal drive. I may also state, without going into unnecessary details, that while the fundamental mechanism which was described by Freud as characteristic of depressions is met in all instances of apparent depression, in some cases of suicide it is not possible to demonstrate that this mechanism is the only determining factor brought into full play in the act. Moreover, as it has already been stated, the clinician observes a number of patients displaying the operation of this mechanism without the suicidal drive being either directly present or effectively operative when present. Hence, the presence or

apparent absence of this mechanism cannot well serve as a reliable diagnostic and prognostic criterion. I shall therefore turn attention to the purely clinical manifestations of some suicidal psychoses and neuroses and try to determine what other features, if any, they possess which could be utilized for diagnostic evaluation.

CLINICAL MANIFESTATIONS OF DIAGNOSTIC VALUE

The woman with a compulsion neurosis, who hanged herself apparently without warning, like many other persons with a similar condition was given to outbursts of anger and was endowed with a bitter and lashing tongue. A few hours before she committed suicide she was her usually surly and aggressively bitter self, which had been familiar to her attendants for many months. However, when the physician failed to pay much sympathetic attention to the "piece of intestine" in the form of a bit of sand cake which she showed to him, she exclaimed that some day she was going to die if no one paid attention to her inner ailments. This was a threat which she carried out impulsively less than three hours later.

This type of suicide appears to be of great clinical importance. It seems to be characteristic of the compulsion neurosis with a suicidal trend. Persons of this type show all the characteristic traits which were described by Freud as anal; they are irritable, persistent, stubborn, cold, contrary, sarcastic, misophobic in various degrees, stingy and spiteful. The well known childish fantasy, "When I am dead they will be sorry," finds its literal expression in their mental life, and their self-murder appears to be an act of aggression against the world, a real act of revenge. The act of suicide appears to have acquired in the fantasy life (mostly unconscious) of these persons a special pleasure value; they libidinize it; it is their supreme expression of hatred. The finer details of their psychic structure have not yet been sufficiently investigated, and the mental processes making such an act possible are still obscure, but there seems to be little doubt that from the clinical point of view they represent a rather treacherous problem, for as has been pointed out, such patients give the impression of constantly ventilating their sadism and one is not infrequently misled into believing that they are too "extraverted" to commit suicide. If they threaten to kill themselves, as this patient did, one is exposed to the danger of falling victim to the common belief that persons who speak of suicide so openly seldom carry out their alleged intentions. As a matter of fact, it is difficult to rid oneself of the impression that spite is a typical cause of suicide and must be seriously taken into account when making a clinical prognostic evaluation in such a case. The presenting symptomatology in cases of this type is rarely that of a depression and almost

always that of a compulsion neurosis with agitation and bitter aggressiveness. When one has the opportunity to study more closely the emotional and instinctual reactions of these patients, one can see more clearly the rôle which a spiteful form of sadism plays in these cases.

Case 7.—A woman in the late thirties, mother of five children, three of whom were living, suffered from a compulsion neurosis of many years' duration. She had been treated by many psychiatrists and had acquired a verbal knowledge of various psychiatric terms, but she failed to improve, because she conveniently converted some of her symptoms into a stubborn pattern which she would not abandon; she was in the habit of consulting a physician with the sole (unconscious) purpose of proving to him that he was unable to cure her. Consciously, she applied herself arduously to the task of being treated; she became devoted to her physician and would not leave him or permit him to leave her even for a day. Her restlessness, feeling of diffuse tension, irritability, querulousness at home and restless, almost ill concealed hatred of people in her social contacts all increased as soon as a long week-end or a brief vacation was suggested or planned by her physician. With many variations this pattern was repeated several times in the course of the illness, which covered a period of almost fifteen years. One day, when she appeared to have been functioning comparatively well for many months, her physician declared that he was going away for a brief holiday; the patient wanted to follow him as she had done with two other physicians in the past. This proved impracticable. The patient responded with an almost instantaneous increase of vague restlessness and became too nervous to play her game of golf, which she usually enjoyed. She stated that she would be unable "to stand it," and she expressed the fear that a panic might develop—a name she gave to her states of tension and restless irritability. The day after the holiday began and one day before the physician left the city, the patient took by mouth a considerable quantity of allylisopropyl barbituric acid with aminopyrine and barbital, and about 1 a. m., as she began to sink under the effect of the drugs, she woke her husband and told him what she had done. She remained unconscious for about seventy-two hours, and before completely regaining consciousness she passed through several hours of hyperkinetic agitation. The first words while she was apparently still unconscious were: "I did it out of spite. I did it out of spite." She did not appear to remember this after she regained complete consciousness.

I shall return to this case a little later in connection with another aspect of the problem. For the present attention is centered on the feature of spite. The purely phenomenologic side of the clinical picture was in the main similar to that displayed by the woman in case 3 who hanged herself. Here again one must emphasize that no matter how incomplete or poorly defined the depressive features in cases of compulsion neurosis may be, agitation, sarcasm and mild panicky states definitely tinged with strong aggression and reactions of hate and spite should be taken as danger signals and possible forerunners of an impulsive suicidal act. Consideration of the unconscious mechanisms and the various infantile libidinous constellations is deliberately omitted at this time, because one cannot subject a person to thorough psycho-

analysis at a glance, and for practical purposes I must confine myself to drawing on the empirical end-results of psychoanalytic therapy. One may thus utilize the psychologic syndromes which reveal themselves and can be observed from the outside, as it were. These purely external diagnostic or prognostic criteria still await full corroboration. Further psychoanalytic studies in such cases will probably permit one later to subject the data to statistical tabulation and thus facilitate and probably corroborate the work on the clinical typology of suicides. However, these preliminary suggestions should serve not only as a guide for further study but as a purely clinical criterion for the serious evaluation of the spite reaction in persons with severe compulsion neuroses.

ARCHAIC TYPES OF REACTION

As has been suggested, one must bear in mind that the spite reaction briefly described is not yet clearly understood. While its external manifestation is easily observed and its dynamic force is incontestable, it appears that, like all other psychologic reactions, it is intimately woven with a number of other contributing factors of various degrees of importance. Of these factors, one is a frequent part of the psychologic structure of suicidal persons and appears to play a major and probably a decisive rôle in a number of suicides. This factor is complex, and it is apparently rooted deeply from both the ontogenetic and the phylogenetic point of view. In order to gain a clearer view of it I shall return to the case of the woman who took allylisopropyl barbituric acid with aminopyrine and barbital.

As psychoanalysis in her case proceeded, the patient gave the increasingly convincing impression that the major part of her unconscious fantasy life was preoccupied with thoughts of extreme resentment toward her mother. No matter how strong her hostility toward her father, she always found extenuating reasons for his behavior, because fundamentally she was deeply attached to him. She even forgave him the alcoholism which had caused her a great deal of concern and pain since she was 14 or 15 years of age. There was nothing she could find in her mother that would awaken in her a warm feeling. There was always hovering over her an almost mysterious, although very vague, idea of death. Her mother was still living. The patient became aware of the intense wishes she had for the death of her mother, but her increasing insight failed to alleviate the panicky sense of the ever present idea of some one's death and her deeply repressed but ever rising awareness that she frequently wished to die for some reason that she was unable to explain. Finally, after many months of work, she recalled what appeared to have been one of the most memorable and traumatic incidents of her life. When she was about 3 years of age her mother became pregnant. Her whole fantasy life then became concentrated around that pregnancy. She hated her mother, and her irritability, captiousness and intractability reached a high degree when a baby brother was born. She hated the newcomer and wished that he were taken away. The baby lived for eight months and died of disease. Memories of that period resuscitated in the course of the psycho-

analysis disclosed that as a girl of about 4 the patient experienced a period of painful anxiety, which never entirely disappeared throughout adult life. Apparently, she passed through the whole gamut of hostile identification with her little brother who soon died. It was this identification with a dead person that seems to have dominated her psychic life and deeply influenced her emotional attitude toward her own children, in relation to whom she harbored at times deep unconscious wishes for death. Evidently, while she was still very young, a little less than 4, her inner conflicts were stimulated to an unusual degree by the pregnancy of her mother, and her sense of guilt was raised to a high degree of tension by the premature death of the hated little brother. When she was 12, just at the time when menstruation began, she experienced another difficult period of jealousy and anxiety and was never able to overcome her intense hatred of her second brother, who was born at that time—a hatred which soon became conscious and under which she labored with the intense ambivalence that is characteristic of compulsion neuroses.

One is justified in stating that the outstanding feature of this patient's psychologic development was the identification with a dead boy just as she somewhat prematurely reached the height of the oedipus conflict. This conflict, tinged with a strong sense of identification with a dead brother, was acutely revived by the birth of a second brother just as she reached another crucial period of life—puberty.

If one compares this psychologic constellation with that of the woman in case 1, one notes the following features:

This patient too as a girl harbored a strong and conscious hatred against her mother, and just at the time when she began to menstruate and her oedipus conflict thus became revived and accentuated the mother died. Her conscious reaction at the time of the mother's death was one of calm and strange lack of regret and sorrow, but a short time afterward she began to show the first signs of hysterical symptoms and a tendency to mild depressive anxiety. In the course of psychoanalysis, fifteen years later, the patient revived and recognized her great attachment to her mother which lay buried under the conscious hatred. It became clear that after the death of the mother the girl passed through a period of severe unconscious mourning, which found expression in the form of her first clinically defined neurotic symptoms. It was at that time that she experienced identification with her dead mother. Here again is a case of identification which was made at the very time that the patient reached a crucial period in psychologic development—in the midst of an acute revival of her oedipus conflicts. This psychologic constellation was clearly reflected in the suicidal act. The decision to kill herself was reached at a time when the patient was apprehensive that her father might marry again. She was alone in the house awaiting the arrival of her father. She perceived a desire to tidy the house; she swept the rooms; she polished the furniture; in short, she assumed the rôle of a conscientious and solicitous housewife, thinking all the time that she wanted to die and that she wished her father to find the house in perfect order. As it has been said, she then put on her best pajamas and her favorite kimono and opened the gas-jets. The outstanding thought at that time was that she wanted to look peaceful and attractive to her father when dead; in other words, in the very act of suicide she acted out her identification with her dead mother.

CASE 8.—A woman, aged 35, who wished to commit suicide by drowning herself with her two little children but who was prevented in time from carrying out her intention, suffered from a series of mild depressions which at times, however, reached considerable severity. The drive to do away with herself appeared at times uncontrollable, and at the time she began psychoanalytic treatment she had been ill clinically for almost nine years. While it would be interesting to review in full the many details in this complex case, which throw considerable light on the origin of the suicidal drives and demonstrate a complex and systematic network of a variety of death wishes, all forming a confluent and cumulative mass of forces that drove the patient to seek death, the clinical aspects in the case will gain in clarity if I limit the review to what appeared the fundamental factor in her psychic economy.

This patient was an only child. When about 6 years of age she discovered a photograph of a little child who was apparently asleep. It was her father's possession and he appeared to cherish it. When she asked him about it he winced and at first gave no answer, but she soon learned that her father had been married to another woman before he married her mother, that that woman had borne him a little son, who died before he reached the age of 1 year, and that the photograph was that of the dead child. Her fantasies took hold of this fact with great avidity. Her father was overheard many times saying that he wished she were a boy, that his first wife's son had died and that about a year after his birth his first wife herself had died in childbirth, the second child being stillborn. They were buried together. The little girl was impressed with these facts at the age of 6, i. e., at the very height of her oedipus problems. Her emotional life was obsessed with this drama; her father loved the dead child more than he loved her; he loved a dead wife and a dead child more than he loved her living mother and his little daughter. An old-fashioned trunk in the house stood at the door of the bedroom of her parents. This trunk was that of the dead wife and was filled with her belongings. The little girl once stole a look into that trunk and discovered a pocketbook with money, dresses and a complete set of baby's clothes—all mementoes which her father cherished. It is impossible, without taking a great deal of space and time, to give a full description of the variety of constellations that grouped themselves with great tenacity in the mind of the little girl around the thought of the dead woman with her dead children. Despite the fact that the patient unconsciously wanted children and although she was happy in her married life, she felt that "for some reason" her children and she should die. The following detail is of decided interest, illustrating the degree of her identification with the dead woman. Her first-born child was a boy, who lived only four hours. Consciously, she reacted to this event with anomalous equanimity; unconsciously, she resented it but never to a great degree. Subsequently she had another child, a girl. She loved this child but wished that it were dead. She did not want any more children, as though she was eager to complete a literal identification with her father's first wife. Her third child, also a girl, was unwanted, but she loved it dearly.

While it is probably a mere coincidence, it should be noted that the patients in all three cases of identification with the dead and in a number of others in my series slept in the bedroom of the parents till very late in childhood. The first patient, who took allylisopropyl barbituric acid with aminopyrine and barbital, slept in the bedroom of her parents until her brother was born; he replaced her till he died, and

then she was returned to her old bed. The second patient, who took illuminating gas, slept in the parental bedroom till about the age of 5, and then she was moved into another room which was separated from the main chamber by a thin partition. The third patient slept till the age of 8 or so in a small room which was a sort of alcove of the bedroom of the parents. The mysterious trunk was near-by, at the door.

Proximity to the bedroom of the parents throughout the formative period of late childhood in many cases in which a great variety of clinical pictures are presented later proves to be a source of unusual stimulation of the oedipus wishes of the child and the conflicts resulting from them. It is not difficult to understand that the stronger these wishes and conflicts the more dangerous the situation is in general from the standpoint of future morbidity and the more dangerous it is in particular in the cases in which a morbid identification takes place at one of the crucial periods of the child's development. The clinical data collected since the publication (1905) of Freud's "Three Contributions to the Theory of Sex" brought ample evidence to corroborate his hypothesis of the psychosexual development of man. There are two pivotal periods in the stages of this development which appear to be crucial, particularly in the life of persons who are liable to be driven to identification with a dead person: These are the oedipus period, the so-called phallic phase of which reaches its height at about the age of 6, and the period of puberty, at which time all the conflicts of the phallic period are revived under the pressure of physiologic maturity. The period between these two phases seems to be silent; it is the so-called period of latency, during which the social adjustment of the child takes precedence over any other aspect of its life and the sexual reactions in the genital sense recede to a great extent into the psychologic background.

In cases of the type just considered, as in those of a predominant spite reaction, it is possible to infer from the clinical picture, trend and anamnesis whether real danger of possible suicide exists if one bears in mind what has been said about identification with a dead person. A history of the death of a person close to the child or a circumstance bringing the theme of death into the actual life of the child at one of the two turning-points of the psychosexual development of the child makes suicide a highly probable outcome, particularly if these events occur with the background of strong and constant stimulation of the child's attachment to one of the parents or to their intimate life (such as sleeping in the parents' bedroom until or beyond the age of 6).

I may add, by way of recapitulation, that evidence of a strong oral anlage or fixation or both, which are not difficult to observe clinically, points to a depressive reaction and also to a greater probability of

suicidal outcome, regardless of the clinical picture—whether that of depression, obsessional neurosis, hysteria or schizophrenia.

One may state, therefore, that a suicidal drive is not dependent on or derived from any traditional clinical entity found in present-day psychiatric nosology; it is to be viewed rather as a reaction of a developmental nature which is universal and common to the mentally sick of all types and probably also to many so-called normal persons. The very universality of the reaction, and particularly of some of its outstanding characteristics, such as oral incorporation, spite and identification with the dead, leads one to suspect that one may be dealing with an archaic form of man's response to his various inner conflicts, and it would prompt one to seek an answer to the problem in the study of primitive races and their reactions leading to suicide. While this thought opens an entirely different, nonclinical approach, it is perhaps of interest to observe several ethnologic parallels which may throw light on the subject.

ETHNOLOGIC DATA

Ethnologic material is still scarce or scattered, and what material is available remains unsystematized and unsubjected to critical study;[6] hence, that which follows represents nothing but illustrative suggestions.

Many legends of primitive races seem to demonstrate some of the mechanisms of suicide which were met in the clinical cases. Thus, in these legends "a sense of being wronged, even though to us the wrong does not seem very serious, is enough to drive the victim to suicide," stated Seligman,[7] by way of preface to the following Melanesian legend of British New Guinea:

> A girl of Southern Massim prefers the Kanioga root to any other food; her mother and aunt twice eat the Kanioga she had collected for herself. When they ask her to come with them to collect more of the desired root, she refuses; then she laid out all her ornaments and finery and put them on, necklaces of shell heads and quill shell armlets, anklets, and a new grass petticoat; then taking her pet dog in her arms she began to sing slowly: "Because of the lily root, because of the lily root I am going." Presently she went into the bush weeping. . . . When the girl came to a lofty wakola tree which was easy to climb, she climbed it, and her mother running up saw her: "Come down, dear, come down," she said, but the girl answered: "No, no, too late," and then looking down she saw a crocodile in the sea close beneath her. . . . She took off her ornaments one by one and threw them to the crocodile who snapped them hungrily; she then threw her dog, and finally her petticoat, until, when naked, she threw herself down and the crocodile took her.

6. The only attempt at a systematic descriptive study of the subject appeared recently by Dr. J. Wisse [Selbstmord und Todesfurcht bei den Naturvölkern, Zutphen, Netherlands, W. J. Thieme, 1933].

7. Seligman, C. G.: Temperament, Conflict and Psychosis in a Stone-Age Population, Brit. J. M. Psychol. **9**:187, 1929.

This legend impresses one as a good symbolic summary of a number of motives and forces which are operative in many suicidal acts of today. It illustrates the ritualistic quality of the act (case 1 of the woman who took illuminating gas, may be compared), and it hints at the deeper dynamics of the wish for death: There is a strong frustration, oral in character, and a deep-seated rejection of further gratification; it is hardly a coincidence that the legend makes the girl die through being eaten by a crocodile; the oral frustration produces a strong reaction of anger (oral sadism) and the culprit dies an oral death.

Another story coming from the Trobiand Islands, related by Malinowski, runs as follows: Kima'i, a boy of 16, had a relationship with his girl cousin, who naturally was forbidden to him by the incest taboo.

> This had been known and generally disapproved of but nothing was done until the girl's discarded lover, who had wanted to marry her, took the initiative. . . . Then, one evening, he insulted the culprit in public, accusing him in the hearing of the whole community of incest and hurling at him a certain expression intolerable to a native. For this there was only one remedy; only one means of escape remained to the unfortunate youth. Next morning he put on his festive attire and ornamentations, climbed a cocoanut palm, and addressed the community, speaking from among the palm leaves and bidding them farewell. He explained the reason for his desperate deed and also launched forth a veiled accusation against the man who had driven him to his death, upon which it became the duty of his clansmen to avenge him. Then he wailed aloud, as is the custom, jumped from the palm some sixty feet high, and was killed on the spot. There followed a fight within the village in which the rival was wounded,[8] and the quarrel was repeated during the funeral.

The deliberateness and the ritualistic quality of this suicide, as of that of the girl of southern Massim, are evident, as is the characteristic method of jumping off a high place. The question of the method used in the act of suicide is of great importance and should present a topic for separate study; it is outside the scope of this communication, however. In regard to the chief points of interest, one can easily see that the motive of incest and the thwarted aggression serve as a legitimate combination for a suicidal outcome; more than that, suicide becomes compulsory under these circumstances. One might state that what is ritualistically compulsory in the primitive community does not disappear from the psychic life of the civilized person; it merely recedes into the background of the unconscious and under proper circumstances of psychologic stress makes its reappearance in the form of irrational impulsiveness, so frequently observed among suicidal persons of the white race. This turning of one's aggression on oneself, legitimatized

8. Malinowski, B.: Crime and Custom in Savage Society, New York, Harcourt, Brace and Company, 1926; quoted by Seligman.[7]

and honored among the primitive and some civilized races, such as those of ancient Rome or present-day Japan, cannot be passed over without comment. It has obviously great sociologic meaning. It performs a social, preservative function: The individual dies because aggression is forbidden to him, but the community takes care of the business of revenge. One wonders to what extent this aspect of social development may not be responsible for the traditional respect many persons have for suicide and for the idealization of his death wish by the suicidal psychotic or neurotic person. At any rate, it is difficult to rid oneself of the impression that many motivations for suicide which are found among patients are only regressive recrudescences of archaic traditions. This appears to be particularly true of the case of suicide in which identification with a dead person appears to predominate, for one finds that the most powerful motivating determinant of suicide among primitive races is the death of a chieftain or of a husband. Thus, the native women of the Solomon Islands follow their dead husbands impulsively: They die by strangulation, which seems to be the oldest method of choice, next to which comes self-immolation on the funeral pyre, practiced by the ancient Hindus and Slavs.

There are a number of motivations for suicide discernible among primitive peoples as atavistic remnants of a still older past, and strikingly enough, the same and, for that matter, all atavistic reactions appear to be still operating in the psychopathologic conditions which come under clinical observation, particularly the compulsion neuroses and schizophrenia.

These sketchy ethnologic illustrations are given here only in order to indicate that there is an impressive parallelism between the traditions of primitive races relating to suicide and the suicides of today. It appears also that not only do typologic impressions find their counterpart among extant primitive races but the clue to the understanding of the deeper determinants and of the causal constellations leading to suicide must be looked for in anthropologic data which are not yet made available to the clinical psychopathologist.

SUMMARY

The problem of early diagnosis of possible suicidal outcomes in patients is of paramount importance in the treatment and prevention of suicidal trends. Such diagnosis is possible on the basis of observations of mental mechanisms and anamnestic data, which become valuable criteria before the suicidal tendency comes out fully in the trend of the patient. Suicides can be differentiated on the basis of the mental mechanisms, this proving that there are many psychologic types of suicide—a point of diagnostic and therapeutic importance.

RELATIONSHIP BETWEEN THE PRESSURE IN THE VEINS ON THE NERVE HEAD AND THE CEREBROSPINAL FLUID PRESSURE

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In 1927 Baurmann [1] reported a method of measuring intracranial venous pressure in man. He compared the pressure in the veins on the head of the optic nerve with the cerebrospinal fluid pressure in nine cases. In these there was a direct relationship between the level of the spinal fluid pressure and his determination of the pressure in the veins on the nerve head (fig. 1). He took as his criterion of the venous pressure the intra-ocular pressure at which the veins on the nerve head first begin to pulsate. The intra-ocular pressure was raised by pressing on the eyeball with an ophthalmodynamometer of the type devised by Magitot, the intra-ocular pressure being measured with a tonometer. All the methods in which venous pulsations are used as a criterion of venous pressure were criticized by Duke-Elder [2] on the ground that venous pulsations in the eye are a complicated phenomenon not directly related to the preexisting intra-ocular venous pressure. Whatever the theoretical objections to the method may be, Baurmann obtained clearcut evidence that the intra-ocular venous pressure, as he measured it, bears a definite relation to the cerebrospinal fluid pressure. It is possible that Baurmann did not measure the true venous pressure, but it is certain that he measured something that is a direct and simple function of the venous pressure, for venous pressure is the only variable involved that can be shown theoretically and experimentally to be directly related to the cerebrospinal fluid pressure.

I have repeated Baurmann's work, using animals, with a slightly different technic as well as a different criterion of the intra-ocular venous pressure. My findings are in entire accord with his.

METHOD

Experiments were conducted on four cats and four dogs. A hollow needle, connected to a reservoir of physiologic solution of sodium chloride, was inserted

From the Eldridge Reeves Johnson Foundation for Medical Physics of the University of Pennsylvania.

1. Baurmann, M.: Weitere Ergebnisse der intrakraniellen Druckmessung mit Hilfe der Netzhautvenenpuls-Beobachtung, Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **46**:96, 1927.

2. Duke-Elder, W. S.: The Venous Pressure of the Eye and Its Relation to Intra-Ocular Pressure, J. Physiol. **61**:409, 1926.

into the anterior chamber. By raising the reservoir, the intra-ocular pressure was raised until a vein on the nerve head showed the first evidence of collapse; i. e., it became paler or narrower. It was believed that the partial collapse of a vessel did not materially alter the general intra-ocular venous pressure and that even if the general venous pressure on the nerve head did alter subsequent to this first collapse, the pressure at which the first collapse occurred, if the pressure was raised rapidly, would be close to the true venous pressure.

The cerebrospinal fluid pressure was varied by means of a pressure bottle connected through a hollow needle with the cisterna magna. Alterations of the cerebrospinal fluid pressure below 18 mm. of mercury did not affect the pressure in the veins on the nerve head. When the cerebrospinal fluid pressure was above 18 mm. of mercury, the intra-ocular venous pressure rose step by step with it, remaining from 2 to 4 mm. of mercury above it. Data obtained from two typical experiments are plotted in figure 1.

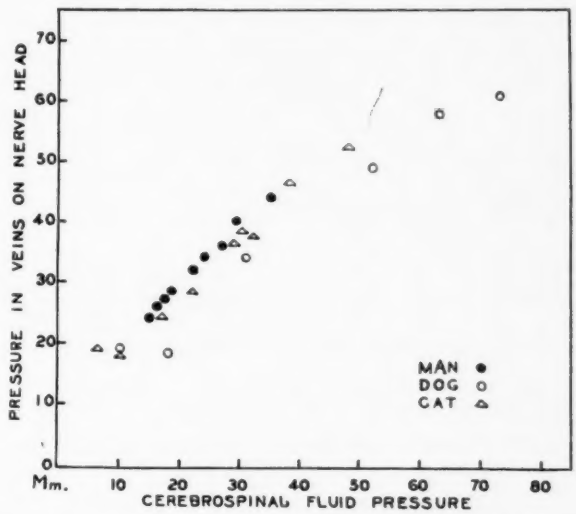

Fig. 1.—Observed relationships between the cerebrospinal fluid pressure and the pressure in the veins on the head of the optic nerve. The data for man are taken from Baurmann's work.

COMMENT

The relation between the cerebrospinal fluid pressure and the intra-ocular venous pressure is not surprising in view of the fact that where the central vein of the retina leaves the optic nerve and traverses the subarachnoid space it is subject to the full hydrostatic pressure of the cerebrospinal fluid. If flow is to continue, the pressure in the central vein must be sufficient to support the wall of the vein against the cerebrospinal fluid pressure. Likewise, the pressure in the veins on the nerve head must be sufficient to support the walls of the veins against the intra-ocular pressure. The venous drainage of the nerve head must

surmount two barriers: (1) the intra-ocular pressure and (2) the cerebrospinal fluid pressure. Unless the basic pressure[3] in the veins on the nerve head is higher than these barriers the pressure in these veins will reflect the pressure in the higher of the barriers. In my experiments the basic venous pressure appears to have been about 18 mm. of mercury; it varied somewhat with respiration. The flattening of the curves as higher pressures were reached is probably to be accounted

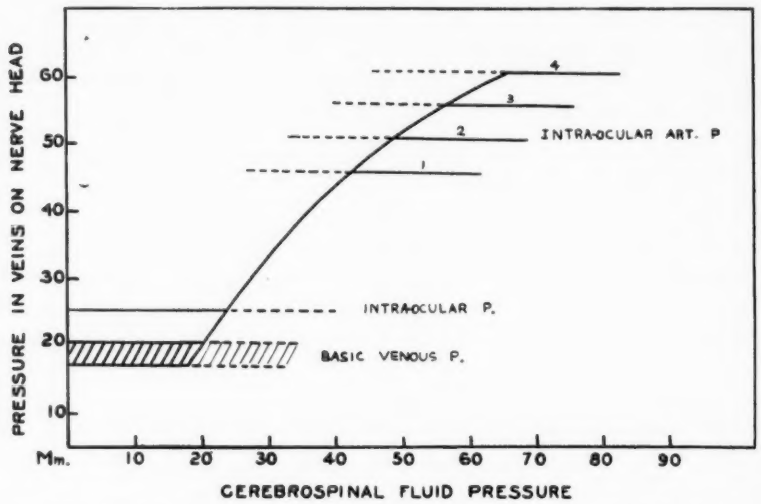

Fig. 2.—The theoretical relationships between the cerebrospinal fluid pressure and the pressure in the veins on the head of the optic nerve. No matter how low the cerebrospinal fluid pressure falls, the pressure in the veins on the nerve head will not go below the basic venous pressure, i. e., the pressure prevailing in these veins when drainage is unobstructed by the intra-ocular pressure or by the cerebrospinal fluid pressure. The basic venous pressure will vary with the phases of respiration; the lower limit of the relationship between it and the cerebrospinal fluid pressure is therefore shown as a broad hatched area. Variations in the cerebrospinal fluid pressure below the intra-ocular pressure will not alter the pressure in the veins on the nerve head. Since normally the intra-ocular pressure is higher than the basic venous pressure, the intra-ocular pressure normally determines the lower limit of the relationship between the cerebrospinal fluid pressure and the pressure in the veins on the nerve head. The upper limit of this relationship is determined by the intra-ocular arterial pressure. As this will rise along with the systemic arterial pressure when the cerebrospinal fluid pressure approaches the level of the arterial pressure, the upper limit will depend on the ability of the animal to raise the arterial pressure to overcome a high cerebrospinal fluid pressure. The upper limit of the relationship is therefore unpredictable and is represented in this figure as a series of possible limits.

3. The basic venous pressure is taken as the lateral pressure which prevails in the veins on the nerve head when their drainage is unobstructed by the intra-ocular pressure or the cerebrospinal fluid pressure.

for by: (1) decrease in the difference in pressure between the veins on the nerve head and the terminal part of the central vein as the flow is slowed and (2) the greater capacity of the wall of a vein to support external pressure as the lumen is narrowed. Final flattening of the upper part of the curve will result, however, when the cerebrospinal fluid pressure rises above the intra-ocular arterial pressure. The pressure in the central vein can then no longer rise; the vein will completely collapse, and further increases in the cerebrospinal fluid pressure will cause no increase in the venous pressure. It will be seen, then, that the relationship between the pressure in the veins on the nerve head is limited below by the basic venous pressure and the intra-ocular pressure and above by the intra-ocular arterial pressure. This is represented graphically in figure 2.

Of practical interest is the fact that this relationship could be taken advantage of in order to determine the presence of a high cerebrospinal fluid pressure when it seemed undesirable to enter the cisterna magna or the lumbar sac. The relationship is of theoretical interest because it shows that the pressure in veins with a truly intracranial drainage varies directly, within broad limits, with the cerebrospinal fluid pressure. This being the case, the relationship illustrated by figure 2 holds not only for the veins on the head of the optic nerve but for all cerebral veins as well, except of course that for these veins the intra-ocular pressure would be eliminated as a determining factor.

CONCLUSIONS

When the cerebrospinal fluid pressure exceeds the intra-ocular pressure and also the basic pressure in the veins on the nerve head, the pressure in these veins varies directly in an almost linear relation with the cerebrospinal fluid pressure until the level of the intra-ocular arterial pressure is reached.

CEREBROSPINAL HYDRODYNAMICS

VII. EFFECTS OF THE INTRAVENOUS INJECTION OF HYPERTONIC SOLUTIONS OF DEXTROSE

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In a previous article of this series [1] it was shown that if after the rapid removal of 35 cc. or more of cerebrospinal fluid from a human subject the pressure of the fluid was allowed to become restored spontaneously to normal and then the same amount of fluid was again drained, the fall in cerebrospinal fluid pressure effected by the second drainage fairly consistently exceeded that produced by the first. This phenomenon was interpreted to indicate that in the interval of time between the first and the second drainage the volume of the ventriculomeningeal spaces had diminished, but whether the diminution was the result of cerebrospinal vascular congestion alone or was in part attributable also to neuraxial edema could not be determined from the data then at hand. In a subsequent report [2] evidence was presented to indicate that hypertonic solutions of dextrose when injected intravenously in sufficient amount and concentration produced a temporary dehydration of the central nervous system which was succeeded within from one to three hours by a secondary period of reactive edema. It is my purpose in the present paper to report a series of experiments in which the procedures employed in the previous studies were combined in an attempt to investigate the effects of the intravenous administration of hypertonic solutions of dextrose on the physiologic and semeiologic disturbances produced in individual patients by the drainage of large amounts of cerebrospinal fluid.

MATERIAL AND METHODS [3]

Ward patients between 20 and 40 years of age who required lumbar drainage for diagnostic purposes but who were free from otherwise determinable organic

From the Baltimore City Hospital and the Phipps Psychiatric Clinic, Baltimore.

1. Masserman, Jules H.: Cerebrospinal Hydrodynamics: IV. Clinical and Experimental Studies, Arch. Neurol. & Psychiat. **32**:523 (Sept.) 1934.

2. Masserman, Jules H.: Effects of Intravenous Administration of Hypertonic Solutions of Dextrose, J. A. M. A. **102**:2084 (June 23) 1934.

3. A more detailed account of the methods employed in these studies will be found in an earlier paper.[1]

disease were chosen for this study. The subject reclined on his left side and relaxed as completely as possible, after which the blood pressure, pulse rate and basic cerebrospinal fluid pressure were determined. A given amount of spinal fluid, which varied in different experiments, was then aspirated within a standard interval of three minutes, and the consequent fall of spinal fluid pressure was recorded. Immediately thereafter various amounts and concentrations of hypertonic solution of dextrose were injected into a cubital vein at the rate of from 10 to 20 cc. per minute. With the subject resting comfortably on his side and the lumbar puncture needle continually in situ, the spinal fluid pressure was read at intervals of five minutes until the original basic tension was restored. At that point spinal fluid to the amount removed in the first drainage was again drained, and the consequent, second, fall in pressure was determined. The spinal fluid pressure was then allowed to reach its initial level a second time, the same amount of fluid was drained once more, and the resultant third drop in pressure was noted. The effects of the intravenous injections and the spinal drainages on the blood pressure and pulse rate of the subjects in the various series were studied, as were the clinical symptoms produced or ameliorated by the respective procedures. The data so obtained were then analyzed according to a statistical technic similar to that employed in the reports of the previous control studies.

RESULTS

Hydrodynamic Effects.—In various series, totaling forty-eight cases, the effects of the intravenous injection of from 50 to 250 cc. of 10 to 35 per cent solutions of dextrose were studied with regard to: (1) the time required for the spontaneous restoration of the cerebrospinal fluid pressure to its original level after the first drainage of spinal fluid, (2) the decrement in the pressure produced by the second drainage of the same amount of fluid, (3) the length of the second interval required for restoration of the basic pressure and (4) the fall in the cerebrospinal fluid pressure after the third and final drainage. An analysis of the data obtained in these preliminary trials, however, showed that the quantities studied did not vary significantly from their respective norms, as determined in preceding control experiments in which dextrose was not administered.[1] It was apparent, therefore, that the administration of comparatively small amounts of dextrose in hypertonic solution produced no constant effect on the dynamics of the cerebrospinal fluid determinable by the methods employed in these studies. When, however, drainages of 35 cc. of spinal fluid were followed by the intravenous injection of 500 cc. of a 35 per cent solution of dextrose, deviations in the hydrodynamic effects from those in the respective controls became evident. The data in these experiments are presented in the accompanying table, series 6 to 10. Analysis of the results in relation to the control constants obtained in the previous experiments (series 1 to 5) shows that:

1. In contrast to the controls (table, series 1 and 2) the mean decrement in the cerebrospinal fluid pressure during the second removal

The Effects of the Intravenous Injection of 500 Cubic Centimeters of a 35 per Cent Solution of Dextrose on the Hydrodynamics of the Cerebrospinal Fluid in Human Beings *

Series No.	No. of Cases	Function	Dextrose	Mean ±P.E.	Series Compared	Difference ±P.E.	Critical Ratio	Statistical Conclusions
1	68	dP_1	No dextrose injected	112.7±3.18	2–1	23.5±4.35	5.15	The mean decrease in the pressure of the cerebrospinal fluid during the second drainage of 35 cc. of spinal fluid exceeded that produced during the first drainage by 23.5 mm. of Ringer's solution
2	41	dP_2		136.2±3.25				
3	20	dP_3		150.0±6.40	3–1	37.3±7.20	5.17	The mean decrease in the cerebrospinal fluid pressure during the third drainage of 35 cc. of fluid exceeded that during the first drainage by 37.3 mm. of Ringer's solution
4	42	Tr_1		115.4±4.05	4–5	26.6±7.54	3.57	The mean time required for the restoration of the pressure of the cerebrospinal fluid after the first drainage of 35 cc. of fluid was 26.6 minutes longer than that required after the second drainage of the same amount of fluid
5	24	Tr_2		88.8±6.36				
6	21	dP_1	500 cc. of a 35 per cent solution of dextrose injected intravenously immediately after the first drainage of 35 cc. of spinal fluid	116.8±5.07	6–1	4.1±6.0	0.70	After the intravenous injection of 500 cc. of a 35 per cent solution of dextrose the mean decrease in spinal fluid pressure during the second drainage of 35 cc. of fluid was not significantly different from that during the first drainage
7	18	dP_2		110.3±5.21	6–7	6.5±7.3	0.90	
8	16	dP_3		152.4±5.90	8–6	35.6±7.8	4.60	After the intravenous injection of 500 cc. of a 35 per cent solution of dextrose the mean decrease in spinal fluid pressure during the third drainage of spinal fluid was 35.6 mm. of Ringer's solution greater than that during the first drainage
9	18	Tr_1		149.8±6.64	9–4	34.4±7.78	4.40	The mean time required for the spontaneous restoration of the original pressure of the spinal fluid after the first drainage of 35 cc. of fluid was prolonged 34.4 minutes above normal by the intravenous injection of 500 cc. of a 35 per cent solution of dextrose
10	16	Tr_2		107.1±5.72	9–10	42.7±8.75	4.90	When 500 cc. of a 35 per cent solution of dextrose was injected intravenously after the first drainage of 35 cc. of fluid the mean time required for the restoration of the cerebrospinal fluid pressure was 42.7 minutes longer than that required for the restoration of the pressure after the second drainage of the same amount of fluid

* Changes in the pressure of the spinal fluid (dP) are given in millimeters of Ringer's solution. The time intervals (Tr) are in minutes. The critical ratios of 3.5 or over are considered statistically significant. dP_1 represents the fall in the cerebrospinal fluid pressure produced by the first drainage of 35 cc. of fluid with the subject in a horizontal position; dP_2 and dP_3, the fall in the cerebrospinal fluid pressure during the second and third drainages, respectively, of 35 cc. of fluid (see text); P.E., the probable error of the corresponding mean of a series or of the mean difference between two series; Tr_1 and Tr_2, the time required for the spontaneous restoration of the initial pressure of the cerebrospinal fluid after the first and second drainages, respectively, of 35 cc. of spinal fluid (see text).

of spinal fluid in the subjects given 500 cc. of a 35 per cent solution of dextrose was not significantly different from the fall in cerebrospinal fluid pressure produced by the first drainage (table, series 6 and 7).

2. Both in the control experiments (table, series 3) and in those in which dextrose was employed (table, series 8) the mean diminution in spinal fluid pressure following the third drainage significantly exceeded that produced by the two preceding drainages.

3. In contrast to the controls (series 4), the mean time required for the restoration of the cerebrospinal fluid pressure after the first drainage in the subjects given dextrose (series 9) was definitely prolonged by about thirty-five minutes.

4. In the series in which dextrose was employed the mean time required for the restoration of the cerebrospinal fluid pressure after the second drainage was shorter by about forty-three minutes than that required for restoration in the first interval (table, series 9 and 10).

Clinical Effects.—In the control study previously reported [1] the combined incidence of various postpuncture complaints (headache, backache, nausea, vertigo and pyrexia) in a total of one hundred and twenty-seven patients from whom 25 cc. or less of spinal fluid was drained was about 34 per cent. During the preliminary trials of the present experiments, 25 cc. or less of spinal fluid was drained from thirty-seven subjects, and this was followed by the intravenous injection of from 50 to 500 cc. of a 15 to 30 per cent solution of dextrose. In these cases the total incidence of postpuncture complaints was about 38 per cent—a difference from that in the controls which is probably not determinative. The administration of greater concentrations of dextrose, however, had a more definitely adverse effect. Thus, of the twenty-one subjects from whom 35 cc. of spinal fluid was drained and who thereafter received 500 cc. of a 35 per cent solution of dextrose intravenously, seventeen (81 per cent) later complained of headache, nausea or vertigo; this contrasts fairly significantly with the incidence of 60 per cent (forty-one of sixty-eight cases) of comparatively less severe complaints in the corresponding series of subjects not given dextrose. It may be inferred, then, that whereas from 50 to 200 cc. of less than a 35 per cent concentration of dextrose when administered intravenously has little if any effect on the incidence of untoward postpuncture semeiology, 500 cc. of a 35 per cent concentration has a definitely adverse influence on the incidence and severity of this syndrome.

Effects on Pulse Rate and Blood Pressure.—As in previous experiments,[1] no significant effects on the pulse rate of the subjects occurred as the result of the intravenous administration of any of the hypertonic solutions employed. The effects on the blood pressure, similarly, were

not sufficiently consistent to warrant detailed presentation, probably because of the combination of experimental procedures employed on each subject. It may be noted, however, that the fairly constant rise in blood pressure that occurred in the series of control subjects approximately two hours after 35 cc. or more of spinal fluid had been rapidly drained [1] occurred also in twenty-three of the thirty-five subjects in the present series who received 500 cc. of a 35 per cent solution of dextrose intravenously after the first drainage of this amount of fluid.

COMMENT

The results of the present investigation can perhaps be best understood in relation to the preceding control studies summarized in the table, series 1 to 5. In these it was shown that after the drainage of 35 cc. of spinal fluid from a human subject the pressure of the cerebrospinal fluid was restored to its original level in a mean time of about one hundred and fifteen minutes (table, series 4). If, then, 35 cc. of spinal fluid was again removed (table, series 2) the decrement in pressure so produced in the majority of cases exceeded by a significant amount the corresponding fall in pressure that occurred during the first drainage. This phenomenon, in view of certain theoretical considerations relevant to cerebrospinal hydrodynamics in the human being, was interpreted to show [1] that at the time of the second drainage of spinal fluid the total volume of the ventriculomeningeal spaces had been encroached on by an increase in the bulk of either or both of the other volumetric components of the dural container: the craniospinal blood vessels or the tissues of the central nervous system. It was then shown that since the alternate removal of spinal fluid and its immediate reinjection caused a progressive increase in the cerebrospinal pressure before there was adequate opportunity for the formation of true edema of the tissues of the central nervous system,[1] part at least of the constriction of the ventriculomeningeal spaces that follows spinal drainage can be attributed to a persistent engorgement of the vessels of the central nervous system. From this it was inferred that some degree of edema of the neuraxis also probably occurs, but proof of this assumption was not forthcoming at the time of the original experiments.

In a later series of investigations to determine the effects of the intravenous administration of hypertonic solutions of dextrose on the pressure of the spinal fluid it was shown that when 500 cc. of a 35 per cent solution of dextrose is administered intravenously to a human subject the cerebrospinal fluid tension, after a transient initial rise, is depressed from 20 to 130 mm. of water below its initial level for a mean time of about three hours, after which it again rises to various heights above normal.

In the light of the previous experimental work of Weed and others,[4] the diminution of the spinal fluid pressure that followed the intravenous administration of dextrose in my experiments was taken to indicate an interval of dehydration and shrinkage of the tissues of the central nervous system which reached its nadir about one hundred minutes after the injection of the solution of dextrose. Conversely, the ensuing rise of cerebrospinal fluid pressure to a level of definite hypertension was taken to correspond with a period of compensatory or reactive edema of the neuraxis.[5]

With these data as a basis the results of the present investigation may be interpreted, and certain further deductions may be made. Thus, since in the present series there was no significant difference between the decrements in spinal fluid pressure produced by the first and the second drainage, respectively, in subjects given 500 cc. of a 35 per cent solution of dextrose (table, series 6 and 7), whereas such a difference did exist in the corresponding control series (table, series 1 and 2), it is probable that in the subjects of the present study the dextrose, exerting its maximum dehydrating action at about the time of the second drainage,[4] counteracted wholly or in part the swelling of the tissue of the central nervous system that would have occurred had the hypertonic

4. Weed, L. H., and McKibben, P. S.: Pressure Changes in the Cerebrospinal Fluid Following Intravenous Injection of Solutions of Various Concentrations, Am. J. Physiol. **48**:512 (May) 1919; Experimental Alteration of Brain Bulk, ibid., p. 531. Haden, R. L.: Therapeutic Application of Alteration of Brain Volume by Injection of Glucose, J. A. M. A. **73**:983 (Sept. 27) 1919. Foley, F. E. B., and Putnam, T. J.: Effect of Salt Ingestion on Cerebro-Spinal Fluid Pressure and Brain Volume, Am. J. Physiol. **53**:464 (Oct.) 1920. Ebaugh, F. G., and Stevenson, G. S.: Measurements of Intracranial Pressure Changes in an Epileptic and Its Experimental Variations, Bull. Johns Hopkins Hosp. **31**:440 (Dec.) 1920. Sachs, E., and Malone, J. Y.: Use of Hypertonic Salt in Experimentally Increased Intracranial Pressure, Am. J. Physiol. **55**:277, 1921. Howe, H. S.: Experimental Studies in the Reduction of the Normal Cerebrospinal Fluid Pressure in Cats, in Dana, C. L., and others: The Human Cerebrospinal Fluid, New York, Paul B. Hoeber, Inc., 1926, vol. 4, p. 220.

5. Fay, T.: Administration of Hypertonic Salt Solutions for Relief of Intracranial Pressure, J. A. M. A. **80**:1445 (May 19) 1923; Comparative Values of Magnesium Sulphate and Sodium Chloride for Relief of Intracranial Tension, ibid. **82**:766 (March 8) 1924. Browder, Jeff: Dangers in the Use of Hypertonic Solutions in Treatment of Brain Injuries, Am. J. Surg. **8**:1213 (June) 1930. Ernst, Max: Untersuchungen über die Wirkung anisotonischer Lösungen auf Gehirn und Liquor, Deutsche Ztschr. f. Chir. **226**:222, 1930. Milles, George, and Hurwitz, Paul: The Effect of Hypertonic Solutions in Cerebrospinal Fluid Pressure, with Special Reference to Secondary Rise and Toxicity, Arch. Surg. **24**:591 (April) 1932. Haug, K.: Klinische und pharmakodynamische Untersuchungen des Liquordrucks vermittelst Dauerdruckmessungen bei Geisteskranken, Arch. f. Psychiat. **97**:189, 1932. Jackson, H.; Kutsunai, T.; Leader, L. O., and Joseph, L. D.: Effect of Hypertonic Dextrose Solutions on Intracranial Pressure in Acute Cranial Injuries, J. A. M. A. **100**:731 (March 11) 1933. Masserman.[2]

solution not been given. This is further borne out by the fact that when the dehydrating effect of the dextrose was expended at the time of the third drainage (approximately four hours after the start of the experiment) the fall in spinal fluid pressure produced by that drainage (table, series 8) exceeded any of the previous comparable observations (table, series 1, 2, 3, 6 and 7), again indicating a marked encroachment of the ventriculosubarachnoid spaces, probably resulting from the enhanced neuraxial hydrosis.

A comparison of the length of the intervals of time required for the restoration of the original pressure of the cerebrospinal fluid after each of the drainages showed results that are also in agreement with this thesis. Thus, the mean interval of restoration after the first drainage of spinal fluid in the subjects given dextrose (table, series 9) significantly exceeded the corresponding interval in the control series (table, series 4), probably indicating a period of retardation in the rate of formation of the cerebrospinal fluid during the period of dehydration of the neuraxis following the administration of dextrose.[6] Conversely, the shortening of the interval required for the restoration of the cerebrospinal fluid pressure after the second drainage may be attributed to a compensatory increase in the rate of formation of spinal fluid, although, as may be seen in comparing series 10 and 5 in the table, this increase was not significantly different from that which occurred after the second drainage of spinal fluid in subjects not given dextrose.

The clinical effects of the intravenous administration of the various solutions of dextrose studied likewise were consistent. As was previously shown,[2] the injection of comparatively small amounts (from 50 to 200 cc.) of weakly hypertonic solutions of dextrose had no consistent effect on the dynamics of the cerebrospinal fluid in human beings; in agreement with this the use of these solutions did not significantly change the number or the character of the postpuncture complaints in the present series. On the other hand, the increase in the incidence and severity of these complaints in subjects given 500 cc. of a 35 per cent solution of dextrose was in keeping with the fact that the intravenous administration of large amounts of strongly hypertonic solutions may cause headache, nausea, vertigo and pain in the peripheral nerves independent of the drainage of spinal fluid. It may be noted relevantly, however, that when moderate amounts of a 20 per cent solution of dextrose are employed after the presumable onset of postpuncture hydrosis of the

6. Foley, F.: Resorption of the Cerebrospinal Fluid by the Choroid Plexuses Under the Influence of Intravenous Injection of Hypertonic Salt Solutions, Arch. Neurol. & Psychiat. **5**:744 (June) 1921. Weed, L. H.: The Absorption of Spinal Fluid into the Venous System, Am. J. Anat. **31**:191 (Jan.) 1923. Forbes, H. H.; Fremont-Smith, F., and Wolff, H. G.: Resorption of Cerebrospinal Fluid Through the Choroid Plexus, Arch. Neurol. & Psychiat. **19**:73 (Jan.) 1928.

neuraxis, a temporary or permanent amelioration of the adverse postpuncture semeiology is obtained,[7] possibly as the result of an advantageous break in the cycle of cerebral congestion and edema induced by the rapid drainage of large amounts of spinal fluid.

SUMMARY AND CONCLUSIONS

Sixty-nine patients were subjected to rapid drainage of spinal fluid and were immediately thereafter given intravenous injections of hypertonic solutions of dextrose in various amounts and concentrations. A study of the subsequent hydrodynamics of the cerebrospinal fluid in these cases confirmed the results of previous control studies and led to the drawing of the following additional conclusions:

1. The constriction of the ventriculosubarachnoid spaces that occurs after the rapid removal of large amounts of spinal fluid is due not only to cerebrospinal vascular congestion but to persistent edema of the tissues of the central nervous system.

2. The intravenous administration of from 50 to 200 cc. of 20 to 35 per cent solutions of dextrose has no determined influence on either postrachicentesic cerebrospinal hydrodynamics or the incidence and severity of postpuncture complaints.

3. In the subjects studied the intravenous administration of hypertonic solutions of dextrose caused no constant effects on the pulse rate or the blood pressure.

4. The intravenous administration of 500 cc. of a 35 per cent solution of dextrose causes a prolongation of the mean period necessary for the restoration of the cerebrospinal fluid pressure to normal and results also in an increased frequency and severity of late postpuncture headache, backache, nausea, vertigo and pyrexia.

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7. Masserman, Jules H.: Intracranial Hydrodynamics: Central Nervous System Shock and Edema Following Rapid Fluid Decompression of the Ventriculo-Subarachnoid Spaces, J. Nerv. & Ment. Dis. **80**:138 (Aug.) 1934.

PERIPHERAL VENOUS BLOOD PRESSURE IN SCHIZOPHRENIC AND IN NORMAL SUBJECTS

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Several deviations from normal standards have been noted in the cardiovascular functions of schizophrenic patients.[1] The venous blood pressure seems to have received little attention. It was decided, therefore, to determine whether there are any differences between the venous blood pressure of schizophrenic patients and that of normal persons and whether peripheral cyanosis bears any detectable relationship to the level of the venous blood pressure.

As regards normal subjects, Moritz and von Tabora,[2] Blumgart[3] and Taylor and his associates[4] reported 10 cm. of water as the upper limit; Villaret and his colleagues[5] placed the upper limit at 13 cm., while Bedford and Wright[6] found that forty-one of forty-seven values for normal subjects ranged between 5 and 15 cm. of water. Rothman[7]

From the Research Service of the Worcester State Hospital and the Memorial Foundation for Neuro-Endocrine Research.

1. (*a*) Hoskins, R. G., and Jellinek, E. M.: The Schizophrenic Personality with Special Regard to Psychologic and Organic Concomitants, A. Research Nerv. & Ment. Dis., Proc. **14**:211, 1933. (*b*) Freeman, H.; Hoskins, R. G., and Sleeper, F. H.: The Blood Pressure in Schizophrenia, Arch. Neurol. & Psychiat. **27**:333 (Feb.) 1932. Freeman, H.: The Arm-to-Carotid Circulation Time in Normal and Schizophrenic Subjects, Psychiatric Quart. **8**:290 (April) 1934. Freeman, H., and Carmichael, H. T.: A Pharmacodynamic Investigation of the Autonomic Nervous System in Schizophrenia: I. Effect of Intravenous Injections of Epinephrine on the Blood Pressure and Pulse Rate, Arch. Neurol. & Psychiat. **33**:342 (Feb.) 1935.

2. Moritz and von Tabora: Deutsches Arch. f. klin. Med. **98**:745, 1910.

3. Blumgart, H. L., and Weiss, S.: Studies on Velocity of Blood Flow, J. Clin. Investigation **4**:15 (April) 1927.

4. Taylor, F. A.; Thomas, A. B., and Schleiter, H. G.: Direct Method for Estimation of Venous Blood Pressure, Proc. Soc. Exper. Biol. & Med. **27**:867 (May) 1930.

5. Villaret, M., and others: Contribution a l'étude clinique de la tension veineuse, Compt. rend. Soc. de biol. **84**:80, 1921.

6. Bedford, D. E., and Wright, S.: Venous Pressure in Normal Individuals, Lancet **2**:106 (July 19) 1924.

7. Rothman, H. R.: Venous Pressure and Its Clinical Significance, J. Oklahoma M. A. **26**:253 (July) 1933.

stated that the normal venous blood pressure is between 4 and 6 cm., while Brown [8] found from 3 to 9 cm. to be the range. Griffith and his associates [9] wrote that any reading above 14 or below 4 cm. of water could be considered pathologic. A moderate degree of variability in the normal venous blood pressure is therefore apparent.

One group of schizophrenic subjects previously investigated showed a mean peripheral venous blood pressure of 13.6 cm. of water.[10] The work of Claude and his colleagues [11] indicated that although normal values are usually obtained in psychotic patients the peripheral venous blood pressure is increased in some persons with dementia praecox, depressed states and dementia paralytica.

METHOD OF INVESTIGATION

The peripheral venous blood pressure can be determined by indirect and by direct methods.[12] The technic adopted in this study was a modification of the direct method of Evans.[13]

The group of subjects studied consisted of fifty schizophrenic patients and twenty-five nonpsychotic subjects free from any demonstrable organic disease. The determinations were made with the subjects in each group under basal and nonbasal conditions. A sufficiently basal condition was considered to obtain if the subject had not had anything by mouth since 6 p. m. of the preceding evening and had rested for one-half hour prior to the determination. For the nonbasal condition, only the one-half hour rest period was required. Basal readings were taken in the morning and nonbasal readings in the afternoon. Personal bias was presumably eliminated in considerable measure, as two observers working independently collected the data. The mean basal readings were within 2 cm. of each other, while the mean nonbasal readings were almost identical. This is a much smaller difference than is noted between the results of independent observers as reported in the literature.

The subject lay flat on a bed which had a minimal amount of sag. He was stripped to the waist to prevent any constriction from clothing. One arm was abducted in the midaxillary line, the forearm was fully extended and a prominent

8. Brown, N. W.: Simple Method of Venous Pressure Determination, Bull. Johns Hopkins Hosp. **29**:93 (April) 1918.

9. Griffith, G. C.; Chamberlain, C. T., and Kitchell, J. R.: A Simplified Apparatus for Direct Venous Pressure Determination Modified from Moritz and von Tabora, Am. J. M. Sc. **187**:371 (March) 1934.

10. Martinengo, V.: La pressione venosa periferica nelle schizofrenie, Schizofrenie **2**:157 (June) 1933.

11. Claude, H.; Targowla, R., and LaMache, A.: Les variations de la tension veineuse au cours de psychopathies, Presse méd. **34**:1193, 1926.

12. Frank and Reh: Ztschr. f. exper. Path. u. Therap. **10**:241, 1912. Frey, A.: Ueber die Bedeutung der Venendruckmessung und so weiter, Deutsches Arch. f. klin. Med. **73**:533, 1902. Gaertner, G.: Die Messung des Druckes im rechten Vorhof, München. med. Wchnschr. **50**:2038, 1903. Moritz and von Tabora.[2] Taylor and others.[4] Villaret and others.[5]

13. Evans, W.: Venous Pressure, New England J. Med. **207**:934 (Nov. 24) 1932.

superficial antecubital vein was selected. The degree of peripheral cyanosis was noted and recorded as absent, slight, moderate or pronounced. The arterial blood pressure and pulse rate were noted. To reduce to a minimum the effect of the venipuncture on the venous pressure, an intradermal wheal was made over the proposed site of puncture with a 2 per cent solution of procaine hydrochloride after the usual preparation of the skin. A short-beveled 20 gage needle was attached to a three way stopcock to which had already been fixed a 10 cc. syringe. An ordinary spinal fluid manometer was utilized to measure the level of the venous pressure. Five cubic centimeters of a 2 per cent solution of potassium citrate, which was used as an anticoagulant, was drawn up into the syringe. When the needle was found to lie freely in the vein the blood pressure cuff was deflated. The spinal fluid manometer was then attached, and while the normal circulation obtained in the vein to overcome venous stasis, the manometer and needle were washed with the potassium citrate solution so that the solution adherent to the walls of the manometer and needle would prevent subsequent clotting. With the manometer empty the blood then rose, and when the upward progress ceased five successive readings in centimeters of blood were taken, with one minute rest periods between readings. After each reading the manometer and needle were flushed with a fresh solution of potassium citrate. A correction of +1.3 cm. was made for the distance between the level of the needle and the zero mark of the graduated scale.

The height, weight and age of each subject were recorded at the conclusion of the determinations. The perpendicular distance between the level of the arm at the site of the venipuncture and the anterior wall of the chest was measured.

RESULTS

The mean basal and nonbasal values of the venous blood pressure for the patients were 11.6 ± 0.46 and 10.9 ± 0.41 cm. of blood, respectively. For the normal group the values were 11.8 ± 0.67 and 11.2 ± 0.50 cm. of blood. Statistically there is no significant difference in these results. As can be seen from the accompanying table, the minima and maxima of the observations were practically the same for the patients and the control subjects (table). The distributions overlapped to a high degree, and the variation from subject to subject was the same in the two groups.

This, however, does not exhaust the possibilities of differences in the venous pressure. One can investigate also the variation which the individual subject may display around his own average level. Applying Fisher's analysis of variance [14] to the data, the following estimates of the average individual variation among five readings in a short time interval (fifteen minutes) were obtained. Under basal conditions the schizophrenic subject showed an average standard deviation of 0.83 cm., while the normal control subject showed an average standard deviation of 1.45 cm. Under nonbasal conditions the values were 0.92 cm. for the schizophrenic and 1.94 cm. for the normal subject. Thus in both

14. Fisher, R. A.: Statistical Methods for Research Workers, ed. 3, London, Oliver & Boyd, 1930.

cases it was found that the schizophrenic patient was less variable than the normal control subject. Testing these differences in mean individual standard deviations for significance, it was found that differences as great or greater than those observed in this study will arise by chance less frequently than once in one hundred trials. It may be said safely that these differences are significant.

More light is thrown on the variability of the venous blood pressure by a correlation of the individual mean values of the basal venous blood pressure with those of the nonbasal venous pressure for a group. All the determinations for each patient were made on the same day. The correlation coefficient for the patients was 0.43, which is fairly low. The correlation coefficient for the normal subjects was not essentially different. These correlations do not invalidate the difference in individual variation between patients and normal subjects, for they were concerned

Mean Values of the Venous Blood Pressure

	No. of Subjects	Maximum	Minimum	Range	Mean and Standard Error of Mean	σ and S.E. σ *
Basal condition						
Mean of five readings						
Patients	50	20.5	6.1	14.4	11.6±0.46	3.2±0.32
Normal subjects	25	17.7	6.3	11.4	11.8±0.67	3.3±0.47
Nonbasal condition						
Mean of five readings						
Patients	50	17.0	4.9	12.1	10.9±0.41	2.9±0.29
Normal subjects	25	18.2	7.5	10.7	11.2±0.50	2.4±0.35

* Standard deviation and standard error of standard deviation.

with the entire group. It means that the patients as a group showed as much variation as the normal subjects.

In our patients under basal and nonbasal conditions, respectively, correlation coefficients of 0.07 and 0.08 were obtained between the venous blood pressure and the systolic blood pressure, and coefficients of 0.17 and 0.22, between the venous pressure and the diastolic blood pressure. Thus these two variables are apparently independent of each other.

It has been noted by numerous observers that the skin of the extremities of schizophrenic patients is more cyanotic than that of normal subjects. Although measurement of the peripheral cyanosis was determined only by crude objective impression and not by the use of color standards, no relationship was indicated between the degree of cyanosis and the level of venous pressure under either basal or nonbasal conditions.

No significant relationship was found between the arm-to-chest distance and the venous pressure. Although the patients as a group were older and weighed less than the normal controls, the results were not rendered invalid, because no significant correlation obtained between age,

weight and the venous pressure. The height of the subjects and the length of the hospitalization of the patients were shown statistically not to be significant. Evidence of seasonal or cyclic trends was not present.

COMMENT

The mean levels of venous blood pressure were similar for schizophrenic patients and normal subjects. The discussion is limited thus to the differences that were found in mean intraindividual variation. Significant differences in such variation between the two groups have been found when other variables were studied. Carmichael and Linder [15] observed that normal subjects individually showed more variability in body temperature than patients and that there was a more constant relationship between the oral and the rectal temperature of schizophrenic patients than between those of normal subjects. This seemed to indicate that independent changes of temperature occur more readily in different parts of the body in the normal person to allow local adaptation.

Hoskins and Jellinek [1a] in a study of systolic and diastolic blood pressure found a more constant relationship of these variables for patients than for normal persons. These facts seem to indicate a tendency of the cardiovascular system of the schizophrenic patient to approach the conditions of a hydrodynamic mechanical system—that is to say, vasomotor responses to changing conditions are relatively sluggish.

The lower degree of mean individual variation in the venous pressure for patients is consistent with these facts and explainable on the hypothesis of the tendency of the cardiovascular system to approach mechanization.

If the veins of schizophrenic patients are considered less capable of reacting to stimuli by dilatation or constriction than those of normal subjects, it follows that the variability of the venous pressure is less. It is probable that the fault lies in the autonomic control system [16] rather than in the blood vessels. There is here, then, further evidence indicating abnormality of response of the autonomic nervous system in persons suffering from schizophrenia.

SUMMARY

Venous blood pressure values were obtained by the direct method under basal as well as nonbasal conditions for fifty schizophrenic patients and twenty-five normal subjects.

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15. Carmichael, H. T., and Linder, F. E.: The Relation Between Oral and Rectal Temperatures in Normal and Schizophrenic Subjects, Am. J. M. Sc. **188**: 68 (July) 1934.

16. Kuntz, A.: The Autonomic Nervous System, Philadelphia, Lea & Febiger, 1929, chap. 7. Howell, W. H.: A Text-Book of Physiology, ed. 12, Philadelphia, W. B. Saunders Company, 1933, pp. 646 and 666.

The mean pressure for patients and normal controls under both basal and nonbasal conditions was 11 cm. of blood.

The mean individual variation of the peripheral venous pressure was less in schizophrenic patients than in normal subjects. Under basal conditions the schizophrenic patient showed an average standard deviation of 0.83 cm., while the normal control showed an average standard deviation of 1.45 cm. Under nonbasal conditions the values were 0.92 and 1.94 cm., respectively.

The peripheral venous pressure did not vary in accordance with the arterial blood pressure.

No significant relationship was indicated between the degree of peripheral cyanosis and the level of the venous blood pressure.

CONCLUSION

This study contributes further evidence that the autonomic reactions of the schizophrenic patient are less than normally facile.

UNILATERAL TRAUMATIC SELECTIVE DEGENERATION OF THE PALLIDUM AND STRIATUM

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It seems strange that a severe trauma of the head could cause during a period of eighteen years following the event a progressive degeneration of the left pallidum and striatum (putamen and caudatum), amounting to complete destruction of these parts, with the corpus Luysi of the same side; yet this is what I have been able to observe in a patient under my care. Some who examined him made a diagnosis of tumor of the brain because of the progressive development of focal symptoms.

The patient was a man who had been thrown from a rapidly moving motorcycle and had received a severe blow on the head. About three years later the pallidostriatal syndrome began to appear and gradually became severe. The patient lived for about eighteen years after the accident. This traumatic syndrome is compared with those of the extrapyramidal system produced by disease. I shall attempt to show that the result of unilateral complete degeneration of the pallidum and striatum, including the corpus Luysi and the zona reticulata of the substantia nigra, in this case was contralateral hypertonia without tremor. In this respect the case would seem to favor the view of Kinnier Wilson that chorea does not depend on striatal lesions.

REPORT OF A CASE

History.—H. C. J., aged 34, was referred to my service by Dr. H. K. Petry from the Warren State Hospital on Feb. 25, 1928. He was discharged on May 22. The history was obtained from the patient and from a letter that he brought from the hospital. The chief complaints were tonic convulsive attacks and an inability to use the right upper and lower limbs properly.

About twelve years previously (1916), having been in perfect health, he was thrown from a motorcycle traveling at from 55 to 60 miles per hour. He was unconscious after the accident for an unknown period and was in a hospital for

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From the Neurological Department of the University of Pennsylvania, D. J. McCarthy Foundation.

Read in abstract at a meeting of the American Neurosurgical Society on June 10, 1935. Microscopic sections of the region of the internal capsule were shown at the meeting of the American Association of Neuropathologists on June 4, 1934.

several months with fractured right upper and lower limbs. After the accident he had a tender area in the midline of the vertex of the head, 2 or 3 inches (from 5 to 8 cm.) in diameter, but the tenderness diminished somewhat in the course of time. Except for this tender area he made a complete recovery which lasted until 1919 or 1920. At that time he began to have tonic spasms of the toes of the right foot. These at first occurred every week or so and during an attack he would take off his shoe and forcibly straighten the toes. The spasms became more frequent, and in 1922 or 1923 he had the four smaller toes of the right foot amputated because of the pain produced by the necessity of walking on the nails of the flexed toes.

At about that time he complained of numbness in the right hand, so that he desired to rub the hand. He also had difficulty in using the hand. Hyperextension of the fingers of this hand developed, and he was unable to flex the fingers into the palm unless aided, although he could open the hand in extending the fingers without difficulty. About 1925 he began to have minor psychic attacks without unconsciousness (euphoria) during which a "streak" went across his eyes. This made him feel so good that he liked to have the attacks, as they seemed "to put pep into him." It was not until about 1927 that he became unconscious during the attacks, which lasted for from five minutes to half an hour. They occurred in series; he would have three or four attacks in a few days, and then a month would pass without any. In the attack he would fall. His wife said that the right upper and lower limbs became rigid and the left limbs were not affected. When the attack was over he would get up and do things of which he later had no recollection. He generally began to undress, and when he regained consciousness he might find himself half undressed and in some other part of the house without knowledge of how he reached that place. The last attack before admission to the University Hospital, six days previously, had occurred while he was going upstairs. He fell down the stairs; after reaching the bottom he stood up and began to undress and then sank to his knees. He had frequently hurt himself in falling.

At the time the numbness developed in the right hand he began to lose the use of the entire right arm, and when he entered the University Hospital this limb was rigid and almost useless. The fingers of the right hand were hyperextended, the ring finger was crossed over the little finger (a position he had assumed at first voluntarily), and the thumb was flexed into the hand. The forearm was less rigid than the hand, and the upper part of the arm was less rigid than the forearm. The right foot became fully extended, and the big toe was flexed over the stumps of the amputated toes.

During 1927 he noticed a distinct impairment of memory. For instance, his wife would tell him to do something, and he would forget it; when an attempt was made to remind him of it he could not remember that he had been told to do anything. This condition had been growing gradually worse.

Speech had been affected for some time. There developed a tendency to stutter, and he could not express himself because of inability to find the words he wanted, even simple words. He had difficulty in spelling; his children would ask him to spell short words, such as "bear," but he could not command the letters he wanted to use. A similar disturbance was associated with attempts to write. He was not word blind or word deaf for ordinary words. When he was given two short articles in a newspaper to read, he read them but had little understanding of what he had read. He had difficulty also in pronouncing long words.

His disposition had changed; he had become irritable and would lose his temper and throw things at the children. This irritability had been growing worse in the few months before admission to the hospital, and his family had found it difficult to live with him. The Wassermann test of the blood and the Wassermann and colloidal gold tests of the spinal fluid gave negative results.

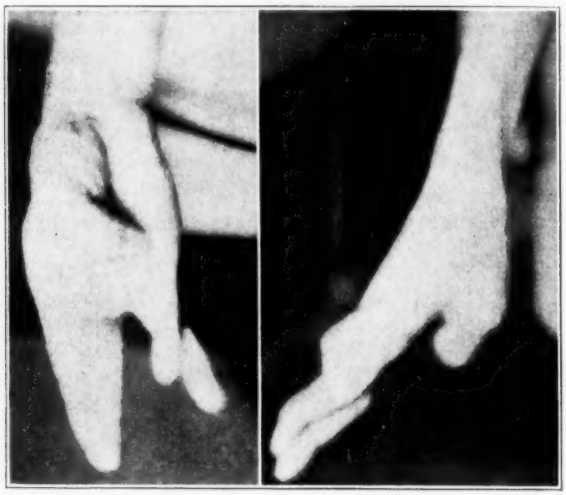

Fig. 1.—Contracture of right hand; the thumb is flexed into the palm, the ring finger is crossed over the little finger, the palm is turned backward in walking and the hand is held behind the body.

Fig. 2.—*A* shows the right foot in full extension; the big toe is flexed over the stump of the other toes. *B* shows absence of paralysis in the right side of the face.

Examination.—On March 7 and 8 I examined the patient. The right foot was in full extension and firmly contractured, so that it was impossible for me to flex the foot dorsally at the ankle. The right leg was not excessively spastic but was moderately so. I could flex it at the knee on the thigh without great difficulty. The achilles tendon reflex on the right was prevented by the contracture;

on the left it was about normal. The patellar reflex was a little exaggerated on the right; on the left it was normal. There was no indication of patellar clonus on the right. The plantar reflex could not be examined on the right because of the contracture of the big toe and the loss of the other toes; there was a distinct flexion of the toes on the left. The entire right leg was moderately atrophied. An attempt to elicit a Babinski reflex on the right produced a violent withdrawal of the foot; the patient stated that the slightest touch on the sole of the right foot produced a sensation of a knife cutting through the sole of the foot, i. e., a violent dysesthesia. This was suggestive of a thalamic lesion. Although no lesion of the thalamus was noted, the function of the thalamus may have been altered by the lesion of the adjoining pallidum and striatum. The entire right leg was only slightly paralyzed. It was impossible for me to prevent him with one hand from flexing the right thigh on the abdomen and the right leg on the thigh, but the resistance to these movements showed that the voluntary motion was not

Fig. 3.—Walking forward as seen from the front and from the rear. Note the position of the right hand.

equal to that of the left leg. It is questionable whether that was indicative of a lesion of the pyramidal tract; it may have been related to the increased tone.

Sensations of position and passive movement could not be determined at the right ankle because of contracture. The right big toe was decidedly impaired in its movement at the metatarsophalangeal articulation. However, slight passive movement at that joint was possible, but passive movement at the articulation of the first with the second phalanx was good, and at that joint there was no sense of position.

Sensation of vibration was not felt at the base of the toes of the right foot but was felt in the right ankle, though less than in the left ankle. The sensations of touch, pain, heat and cold seemed to be lost in the right leg below the knee but began to be felt at the knee, though not with the same acuteness as on the left side. There was a moderate degree of these sensations in the right thigh and in the right side of the trunk. Pressure sensation produced by the point of a pencil was

promptly recognized throughout the right leg, even in the foot, where the sensations of touch, pain, heat and cold appeared to be lost. The sensations of touch, pain, heat and cold were impaired in the right arm. These sensations were partially impaired in the right side of the face. Pressure sensation was impaired in the right arm but was correctly localized after a moderate degree of pressure even in the right hand. There may have been slight impairment of pressure sensa-

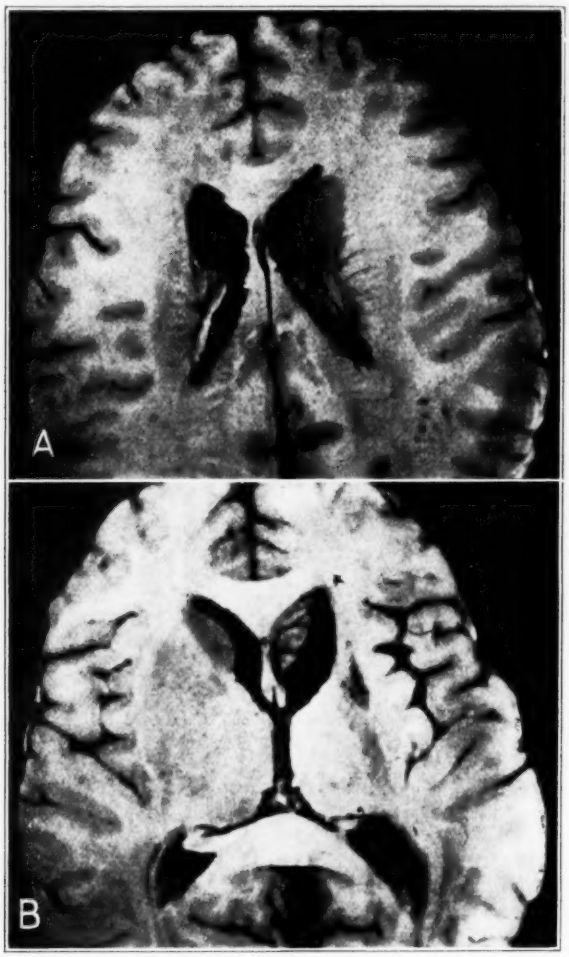

Fig. 4.—*A*, horizontal section seen from above. The anterior horn of the left lateral ventricle is larger, owing to destruction of the head of the caudatum. *B*, same section as *A* but seen from the lower level.

tion in the right side of the face. The marked contractures of the fingers of the right hand interfered greatly with sensations of passive movement and position; these sensations were impaired at the wrist but were acute at the elbow. Contracture was present at the right wrist. The sensation of vibration was not recognized in the right arm until the elbow was reached, and there this sensation was

impaired. The right hand was markedly contractured; the fingers were in extension; the ring finger was held over the little finger as the result of an intentional position, and there was partial flexion of the terminal phalanx of each finger. The thumb was firmly flexed into the palm of the hand. In walking the right leg was swung from the hip.

This man was examined by several members of the neurologic staff and it was found that sensory disturbances varied considerably in the right side of the body in the different examinations; they appeared to be influenced even by suggestion, so that hysteria was considered as a possible condition.

The right hand was contractured firmly in pronation at the wrist but not at the elbow. The power in the right arm was excellent. I could not prevent its voluntary movement, but it did not appear to be as great as that in the left arm. The right side of the face was well innervated, but the right corner of the mouth in showing the teeth or in separate movements was not as well drawn up as the left corner. The furrows about the right eye when the lids were firmly closed and the wrinkles of the right side of the forehead were not as marked as on the

Fig. 5.—Microscopic section from the same level as the section shown in figure 4 *B*. Note the integrity of the posterior limb of the internal capsule. There is possibly some impairment of the anterior limb of this capsule.

left side. The right arm was not atrophied. In walking the right arm was carried behind the back with the palm of the hand outward.

The spasms were epileptiform but purely tonic and were of the character of spasms which I have described as due to subcortical epilepsy. They were confined to the limbs of the right side.

Diagnosis.—I regarded this man's condition as one of subcortical epilepsy, possibly the result of the injury sustained from the motorcycle accident, with a lesion in the left striatum and pallidum. The symptoms were those of involvement of the extrapyramidal system.

Autopsy.—The man died on March 6, 1934. The brain was hardened in a solution of formaldehyde U. S. P. made neutral and sodium chloride and sent to me by Dr. Darling and Dr. Eaton, of the Warren State Hospital, on April 1, 1934. It was received in excellent condition. The cause of death was acute pulmonary tuberculosis, with laryngitis and peritonitis.

In the left cerebral hemisphere Broca's area was somewhat retracted, as though there were some subcortical lesion in that region. It was different in its

development from that of the corresponding area in the right cerebral hemisphere. A horizontal cut made according to the method of Dejerine, passing through the upper part of the internal capsule, showed a scar at the outer side of the knee of the left internal capsule. This affected slightly the anterior limb of the internal capsule but not the posterior limb. It was situated in the pallidum and the putamen. The anterior horn of the left lateral ventricle was greatly distended and much larger than that of the right lateral ventricle. This was because of the complete disappearance of the head of the caudatum. Another horizontal section made a little more than ¼ inch (0.6 cm.) below the previous one showed complete destruction of the entire lenticular nucleus.

Microscopically, sections at this level, stained with Weil's stain and Weigert's hematoxylin stain showed some loss of the nerve fibers in the anterior limb of the internal capsule, but the posterior limb was intact. The pallidum and putamen were entirely destroyed except for a small almond-

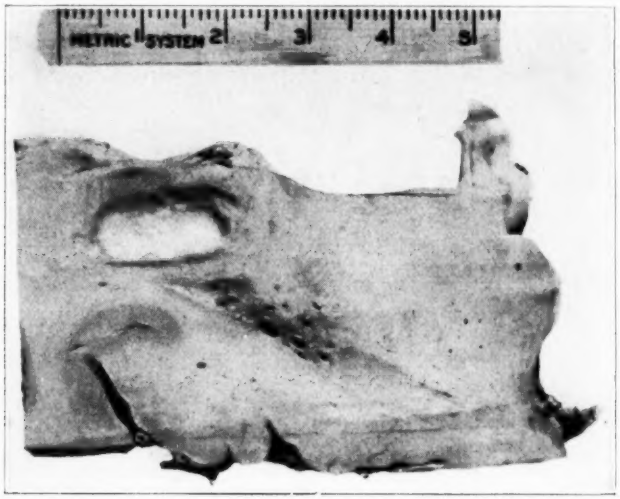

Fig. 6.—Cavity formed by the disappearance of the tip of the head of the left caudatum. This section was from a level below that of figure 4.

shaped area at the posterior end, which was all that remained of these structures. This contained medullated fibers and possibly some degenerated nerve cells. The remainder of this area was occupied by scar tissue of loose glial structure, containing blood vessels and numerous small, round nuclei. There was considerable blood pigment here. The optic thalamus presented a normal structure, with medullated nerve fibers and nerve cells. The caudatum had entirely disappeared, leaving no trace of its former existence. A slight concavity at the inner side of the anterior limb of the internal capsule corresponded to the shape of the head of the destroyed caudatum; and at a lower level, where this structure ends in the brain substance, an empty cavity, exactly corresponding to the termination of the head, was seen. There was no evidence of the fibers belonging to the tail of the caudatum.

Sections from the inner and top portions of the left paracentral cortex, stained with cresyl violet, showed no distinct pathologic changes. The Betz cells were

numerous and normal. The Weigert hematoxylin stain showed normal nerve fibers. The same was true of sections taken from the right paracentral cortex.

In the inner portion of the left paracentral lobule a blood vessel was observed surrounded by what appeared to be hemosiderin granules. This was in the part corresponding to the tender area which had been noted during life in the scalp in the midline region of the vertex. At Dr. Lucke's suggestion sections from this portion of the paracentral lobule and from the degenerated left pallidum and putamen, which contained many similar granules in the scar tissue, were stained for Turnbull's prussian blue reaction for iron. The results were typical in the latter sections but not so satisfactory in the sections from the paracentral lobule, as probably only a part of the vessel mentioned was contained in them. Sections treated with sodium hydroxide (4 Gm. to the liter) for an hour to remove formaldehyde precipitate showed that the results were characteristic for iron; therefore, the granules were hemosiderin from a hemorrhage which had occurred about eighteen years previously.

The pigmented cells of the zona compacta of the left substantia nigra were numerous and appeared normal, but there was much loss of the cells of the zona reticulata. The left corpus subthalamicum had disappeared as a definite formation, although some cells were present which may have belonged to that structure.

The nerve cells and medullated nerve fibers of each nucleus ruber were numerous and normal, and the two nuclei were of about equal size. The cells and nerve fibers of the right and left dentate nuclei were normal. A section from the right cerebellar cortex was normal.

The two anterior pyramids of the medulla oblongata were about the same size, and both stained well by the Weigert hematoxylin method, showing that the posterior limb of the internal capsule was not degenerated.

COMMENT

The extraordinarily selective character of the degeneration of the pallidum and striatum is not easy to explain. At first an attempt was made to attribute it to vascular distribution, but this proved unsuccessful. According to Beevor,[1] the head of the caudatum is supplied by the anterior and middle cerebral arteries; the distribution varies in degree in different brains and is similar to that of the anterior part of the lenticular nucleus. These two arteries supply also the anterior segment of the internal capsule. The superior horizontal part of the caudatum is supplied by the middle cerebral artery. The tail, or surcingle, in its posterior part is nourished by the posterior cerebral artery as far posteriorly as the upper end of the descending cornu of the lateral ventricle. From this point forward to its termination in the nucleus amygdalae it is supplied by the anterior choroid artery.

The external third segment (putamen) of the lenticular nucleus is supplied mostly by the middle cerebral artery; the anterior inferior part in some cases is supplied by the anterior cerebral artery. The anterior choroid artery may supply the posterior and inferior parts of this seg-

1. Beevor, Charles E.: On the Distribution of the Different Arteries Supplying the Human Brain, Phil. Tr. London, s. B. **200**:1, 1909.

ment. The middle, or second, segment of the lenticular nucleus is supplied chiefly by the anterior and middle cerebral arteries and also in some cases by the anterior choroid artery. The internal, or first, segment is supplied largely by the anterior choroid artery; in less degree, by the posterior communicating artery or the anterior cerebral artery.

From this description by Beevor it appears unlikely that all parts of the pallidum and striatum could be selectively destroyed by thrombosis or hemorrhage, leaving the surrounding tissue intact.

Cornelia de Lange [2] possibly gave a clue to the complete disappearance of the caudatum in my case which may be acceptable. She described the brain of a child who had congenital hypertrophy of the muscles of the body. Numerous cavities, some of them visible to the naked eye, were seen throughout the brain. Some were empty and lined with gitter cells; others were filled with fluid that stained red with eosin; a zone of thickened glia often was around the cavities, and farther away was the status spongiosus, resembling that seen in the degeneration of tracts in cases of pernicious anemia. The condition was described as aseptic necrosis or porencephaly. After death, within the first year following traumatic hemorrhage of the brain, extravasation in the process of being absorbed, or brown spots or deposits of pigment may be noted. It is possible that contusions, for instance, those resulting from a series of small concussions of the brain, may cause porencephaly or aseptic necrosis. She referred to extensive studies made by Spatz, Siegmund and Schwartz of the pathologic histology of porencephaly caused by injuries at birth. Spatz concluded from investigations on the spinal cord of new-born rabbits that the specific reaction of the nervous system to trauma is the early appearance of necrosis and cavities, without the formation of scars.

I have seen necrosis of the pallidum following carbon monoxide poisoning; the condition is well known. Spatz has been quoted as saying that the pallidum is especially susceptible to toxic conditions; it is possible that the striatum also is susceptible to such conditions.

The case which I present affords some opportunity for comment as to the functions of the pallidum and striatum. The complete destruction of these structures on one side only, without any evidence of disease, is an extraordinary event. Much dispute has arisen regarding the symptomatology of such lesions, especially as the lesions have usually been bilateral and often associated with those of other parts of the brain and even with a remote organ, such as the liver.

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2. de Lange, Cornelia: Congenital Hypertrophy of the Muscles, Extrapyramidal Motor Disturbances and Mental Deficiency, Am. J. Dis. Child. **48**:243 (Aug.) 1934.

The Vogts [3] in 1919 stated that there are (1) a definitely determined pallidothalamic tract to the ventral portion of the medial thalamic area, (2) an ipsilateral and a contralateral pallidorubral tract and (3) a pallidum-corpus subthalamicum tract. Tract 1 might cause a loss of fibers to the optic thalamus, which after eighteen years might be difficult to recognize; the optic thalamus in my case seemed to be intact. Tract 3 might explain the disappearance of the left corpus subthalamicum in my case. These authors also asserted that the striatal syndrome may be unilateral, but the pallidal syndrome is never unilateral, since the pallidum sends fiber tracts to both sides of the central nervous system, as they indicated in describing tract 2.

The symptoms in my case were entirely unilateral, without any evidence of pallidal disturbance on the left side of the body. The hypertonia of the right side of the body in the upper and lower limbs was pronounced and was associated with contractures of the hand and foot. There were no involuntary movement and no form of tremor in the right upper and lower limbs, notwithstanding the disappearance of the left corpus Luysi.

Other writers have referred to the effect of trauma on patients belonging to the group under discussion. Lotmar [4] paid especial attention to it. He said that injury at birth has been accepted as a cause of status marmoratus of the striatum by Schwartz, and Lotmar also accepted it. He did not accept the case of Bostroem and Spatz as one of an endogenous process, as did the authors, because of athetosis dating from birth, idiocy and the indicated status marmoratus of the striatum, because in Schwartz' case there was selective birth injury of the dentatum of one side with other birth injuries of the cerebrum. Lotmar expressed the opinion that in Bostroem and Spatz' case the condition was the result of birth trauma, although no mention was made of such an injury by the authors. He stated that birth trauma has an etiologic relation to the status marmoratus of the striatum. He did not elaborate on the manner in which birth trauma produces this effect.

Fünfgeld,[5] writing on this subject, said that the Vogts described the cases of two patients whose symptoms began in earliest childhood with athetosis and epileptic attacks, with increasing hypertonia until death. The authors noted a peculiar disappearance of fibers in the pallidum and

3. Vogt, Cécile, and Vogt, Oscar: Zur Kenntnis der pathologischen Veränderungen des Striatum und des Pallidum und zur Pathophysiologie der dabei auftretenden Krankheitserscheinungen, Sitzungsb. d. Heidelberg. Akad. d. Wissensch., 1919. (This was lent to me by Dr. E. Spiegel.)

4. Lotmar, F.: Das extrapyramidal-motorische System und seine Erkrankungen, Fortschr. d. Neurol., Psychiat. **3**:245, 1931.

5. Fünfgeld, E.: Zur Klinik und Pathologie frühkindlicher, das striäre System bevorzugender Hirnerkrankungen, J. f. Psychol. u. Neurol. **40**:85, 1929.

striatum which they described as a status dysmyelinatus. They included a case described by O. Fischer and one by Rothmann in their group. Since then other cases have been reported by Filimonoff and Onari and especially by Spatz and Hallervorden, who included their cases of familial disease of the pallidum and substantia nigra in the class described by the Vogts. Kononova later added another case, and Fünfgeld added two more cases. In the first case Fünfgeld observed marked alteration of the pallidum and striatum, including the cerebral cortex, the thalamus and the substantia nigra. He said that difficult labor occurred in his case, in the two cases of the Vogts and in the case of Onari. He explained his views regarding the manner in which this acts. He attributed a pathogenic factor to it, not by traumatic hemorrhage but possibly by impaired nutrition during prolonged labor or by some peculiarities in the maternal or infantile organism which in some unknown manner delayed the birth. He noted a peculiar deposit from the tissue fluid in the pallidum and putamen but especially a loss of myelin sheaths in these parts.

The number of disorders attributed to the areas affected in my case is great. Lotmar included as the diseases of the extrapyramidal motor system Huntington's chorea, chorea minor, hemiballismus, myoclonia, myoclonic epilepsy, athetosis, status dysmyelinatus of the pallidum (Hallervorden-Spatz disease), the dentatum group of double athetosis with degeneration of the anterior cerebellar peduncles, torsion spasm, torticollis spastica and organic tics. Why tremor exists in one case and athetosis in another he did not attempt to explain. One may add to this list Hunt's juvenile paralysis agitans and Winkelman's progressive pallidal degeneration.

Kihn [6] made a careful review of the work on chorea. He stated that it is not possible in Huntington's chorea to regard the abnormal condition of the striatum as the cause of the disease; one may say only that the neostriatum in Huntington's chorea belongs to the areas of the brain which are most affected. The pallidum, thalamus, corpus Luysi, cerebral cortex and white matter may be greatly involved. There is no selective degeneration of the small cells of the striatum, and it is possible to speak only of progressive degeneration of the ganglion cells in the striatum and pallidum, although the caudatum and putamen seem to be more altered than the pallidum. In speaking of other forms of chorea he said that focal lesions of the caudatum and putamen show a definite relation to chorea, but the rôle of the pallidum is uncertain, as isolated lesions in it have not been noted in cases of chorea. Cortical chorea

6. Kihn, B.: Probleme der Choreaforschung, Nervenarzt **6**:505 (Oct. 15) 1933.

has not been established, as Kinnier Wilson held, and he looked on the striatal theory for chorea as unfounded (1930).

Chorea has been observed with lesions of the cerebral cortex, cerebellum, anterior cerebellar peduncles, nucleus ruber, substantia nigra, corpora quadrigemina, subthalamic region, corpus Luysi, ansa lenticularis, thalamus, striatum and pallidum, i. e., almost any part of the brain. The striate system has no direct cortical connection. The first series of neurons is from the prefrontal cortex and the precentral convolution to the anterior medial nucleus of the thalamus. From this nucleus the second series of neurons goes to the pallidum, caudatum and putamen. The interstriate neurons, as association tracts in the neostriatum, form the third system of neurons. The fourth series of neurons is from the caudatum and putamen to the pallidum. From here there is a connection with the thalamus and back to the cortex. There is also a connection from one pallidum to the other and to the corpus Luysi and other connections to lower parts, mostly questionable. There may be a connection with the pyramidal tract.

As a result of the investigation already carried out it seems to me that the different clinical manifestations of extrapyramidal disease must depend largely on different groupings of lesions affecting this system. The lesions produced by trauma, which I am now reporting, are similar to the bilateral lesions I reported in a case of acquired double athetosis in 1920.[7]

Zingerle [8] has reported a peculiar disturbance of function resulting from a lesion of the anterior limb of the internal capsule, the tractus frontocerebellaris. His cases were purely clinical. His first case was one in which a man while walking suddenly became hypertonic in the left leg and soon also in the left arm; the body was drawn to the left, but he could move these spastic limbs, although he could not turn to the right while lying down because of the spasticity of the left side. Later he was not able to perform the associated movements of the left limbs with the right in standing up, walking, climbing steps or turning, but he was not paralyzed. This complex Zingerle attributed to a lesion of the frontal lobe. He quoted extensively from the literature to support his diagnosis. Pötzl expressed the belief that the four foot gait of the animal has been changed to the upright gait of man through the action of the frontal lobe. Gerstman has described the motor disturbance in disease of the frontal lobe as frontal astasia-abasia. Such symptoms as these are attributed by Zingerle to a lesion of the tractus frontocerebellaris, which he said has been regarded by von Monakow, Jacob,

7. Spiller, W. G.: Acquired Double Athetosis (Dystonia Lenticularis), Arch. Neurol. & Psychiat. **4**:370 (Oct.) 1920.

8. Zingerle, H.: Ztschr. f. d. ges. Neurol. u. Psychiat. **149**:737, 1934.

Pötzl and Hoff as one of the most important parts of the motor system. It arises in the anterior basal and medial cortex of the prefrontal area as far as the operculum. It is somewhat difficult from these statements to distinguish the syndrome produced by a lesion of the tractus fronto-cerebellaris from that produced by a lesion of the striatum and pallidum.

The anterior limb of the internal capsule was regarded by Dejerine as having no part in the inner portion of the foot of the cerebral peduncle, which in his opinion does not contain cerebellar fibers. He said that the anterior limb of the capsule passes to the optic thalamus. The anterior limb was not intact in my case, but the symptoms mentioned by Zingerle, apart from the hypertonicity, were not present.

The photographs shown in figures 1 to 4, inclusive, are enlargements from a moving picture film taken by Dr. Ornsteen.

EPILEPTIFORM ATTACKS IN CASES OF GLIOMA OF THE CEREBRAL HEMISPHERES

RELATION TO THE LOCATION AND HISTOLOGIC TYPE OF THE GLIOMA

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Much has already been written concerning epileptiform seizures in cases of intracranial neoplasm. In most reports the frequency and type of epileptiform attacks are discussed in reference to the location of the tumor (Parker, Gibbs); little or no attention has been paid to the histologic type of the lesion. This kind of approach has been attempted only by R. Groff, in a recent contribution discussing epileptic attacks in cases of meningioma. Of late it has been recognized that lesions situated in certain cerebral areas do not always produce a constant neurologic syndrome and that the clinical signs may vary in accordance with the histologic nature of the disease (Bailey and Cushing). From this point of view I have analyzed the frequency and type of epileptiform attacks in cases of glioma. Special consideration has been given to the clinical symptomatology of the various subgroups of glioma.

MATERIAL

This paper is based on a study of 300 verified cases of glioma of the cerebral hemispheres with sufficiently detailed clinical records. In 57 cases personal observations were made at the clinics of Prof. P. Schuster and M. Borchardt, Berlin; Dr. Harvey Cushing, Boston; Dr. M. M. Peet, Ann Arbor, Mich., and Dr. Laruelle, Brussels. The remainder of the cases have been taken from reports in the current literature.

In order to minimize the statistical error it seemed advisable to study only cases of glioma of the cerebral hemispheres, excluding all instances in which the tumor was confined to the diencephalic region, basal ganglia, brain stem or cerebellum, since it is well known that a telencephalic lesion is more likely to cause epileptiform attacks. Furthermore, as the different types of glioma occur in different frequency in the supratentorial and infratentorial regions, the aforementioned division became necessary.

DEFINITION OF EPILEPTIFORM ATTACK

Before any detailed analysis of the problem is attempted it is necessary to define the term epileptiform attack as it will be used in this paper. Whereas Parker included in his report only cases in which there

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were major epileptic seizures, I think that such a definition rather arbitrarily rules out a large number of observations. With Penfield, I consider as epileptiform seizures not only major attacks (generalized or focal) but any other manifestation of neuronal discharge, such as the different types of petit mal attacks and the paroxysmal epileptic equivalents (uncinate attacks, certain types of so-called optic and auditory hallucinations, dreamy states and certain phenomena of the vegetative sphere). Attacks of unconsciousness without other signs, fainting attacks, paroxysmal tremors and myoclonias were not conconsidered.

Epileptiform seizures result from a combined action of many factors; there exist, for example, general causes, such as constitutional predisposition and a disturbed physicochemical equilibrium of the patient at the time of the attack. In this study, however, I shall consider only the local factors, such as (1) the position of the glioma and (2) its histologic structure.

RELATIONSHIP BETWEEN EPILEPTIFORM ATTACKS AND LOCATION OF THE GLIOMA

Neurologic symptoms of tumor of the brain must be evaluated with the utmost caution as bearing on the problem of cerebral localization. Furthermore, in cases of glioma one frequently faces difficulties in determining exactly the gross anatomic site of the tumor. Most of the extracerebral neoplasms are clearly demarcated, and their location can be defined with comparative ease. Glioma, however, often presents no definite line of demarcation. Even such gliomas as appear grossly to be well delimited may prove to be diffusely infiltrative microscopically (e. g., oligodendroglioma). In addition, the changes of the neighboring noninfiltrated brain tissue must be considered, as they also may play an important rôle in the production of epileptiform attacks. Of course, extension of a tumor as determined only by operation is much less trustworthy than when verified by autopsy.

In spite of these sources of error, the figures in table 1 fairly approximate the accepted rules of cerebral localization. The incidence of epileptiform attacks and equivalents happens to be very high (53 per cent) in the glioma material. For this reason the percentages for most of the cerebral areas are much larger than those cited by Parker and Gibbs. In my series, glioma of the frontal,[1] parietal and temporal lobes was associated with epilepsy in about 50 per cent of the cases, and glioma of the frontoparietal (rolandic and postrolandic) area, in 79.2 per cent, whereas the frequency of attacks decreased considerably in the

1. In this paper the term frontal will be used only for the region anterior to the precentral gyrus. Lesions occupying chiefly the rolandic and postrolandic gyrus will be listed as gliomas of the frontoparietal region.

TABLE 1.—*Frequency and Type of Epilepsy in Relation to Location of the Tumor*

Region	Total Number	With Epilepsy		Type of Epilepsy													
				General		Petit Mal		Jacksonian Attacks				Uncinate Attacks		Visual Attacks		Acoustic Attacks	
								Motor		Sensory							
		Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent
Frontal	73	38	52.1	24	49	5	10	16	33	2	4	2	4	..	..	..	..
Frontoparietal	48	38	79.2	11	21	5	9	26	50	10	19	1	1	..	..	..	..
Frontotemporal	19	8	42.2	5	33	4	27	1	7	3	20	1	7	1	7	..	..
Temporal	65	33	50.8	18	39	7	14	8	16	3	6	9	18	4	7	..	..
Parietal	26	13	50.0	5	24	..	..	9	43	7	33	..	..	..	..	..	..
Parietotemporal	11	6	54.5	3	43	..	..	2	29	1	14	1	14	..	..	..	..
Frontoparietotemporal*	4	3	75.0	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Occipital	10	2	20.0	1	33	..	..	..	..	1	33	..	..	1	33	..	..
Temporo-occipital	15	6	40.0	2	18	2	18	..	..	..	..	2	18	4	37	1	9
Parieto-occipital	16	6	37.5	3	43	..	..	2	29	1	14	1	14	..	..	..	..
Frontoparieto-occipital*	2	1	50.0	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Temporoparieto-occipital*	6	2	33.3	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Whole hemisphere*	3	2	66.6	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Fornix and corpus callosum*	2	1	50.0	..	..	..	..	..	..	..	..	..	..	..	..	..	.

* No figures are given for the different types of attacks because of the small number of cases. Furthermore, as most of these regional groups involved large parts of the hemisphere, a classification of attacks seemed to be without value for localizing purposes. Note that the percentages in columns 5, 7, 9, etc. refer to the total number of attacks; this was done because many patients had more than one type of attack.

cases of glioma of the posterior part of the hemisphere, epilepsy being associated with glioma of the parieto-occipital region in 37.5 per cent of the cases and with glioma of the occipital region in 20 per cent. Much more important is the relationship of a certain type of epileptic attack to a given area of the brain. Here, again, my findings correspond grossly with the results obtained by Foerster and Penfield on the basis of a different approach (experimental electrical stimulation of the cerebral cortex in cases of epilepsy not associated with a neoplasm).

1. *Generalized Epileptic Seizures (Grand Mal).*—Only generalized attacks, without localizing evidence, are listed in this group. Possibly their number is too high in this series, as discrete localizing signs in the beginning of the attacks might have been overlooked.[2] It should also be remembered that any focal attack may become generalized with increasing severity and spread of the discharge. Such generalized seizures without focal signs occurred most frequently in cases of glioma of the frontal region (49 per cent of attacks; table 1) and less in cases of glioma of the temporal region (39 per cent of attacks), and a still lower percentage was found in cases of glioma of the fronto-parietal region, although the latter was associated with the highest total incidence of epilepsy. On the other hand, grand mal was comparatively common in cases of tumor of the occipital and parieto-occipital regions.

2. *Petit Mal.*—These attacks are not frequent in cases of tumor of the brain. They were observed chiefly in cases of glioma of the temporal and frontal lobes or in the areas adjacent to these parts (frontoparietal and temporo-occipital). The frequency reached the highest percentage in cases of glioma in or near the temporal region (in cases of glioma of the temporal region, 14 per cent; of the temporofrontal region, 27 per cent; of the temporo-occipital region, 18 per cent). In other words, petit mal attacks seem to occur frequently in association with lesions located in or near the area supplied by the middle cerebral artery. While petit mal represents a rather ill defined type clinically, it is remarkable how often the speech mechanism is disturbed in these seizures, which in some cases may be nothing more than a short period of aphasia (MacRobert and Hens). In other instances the petit mal attacks not only consist of, or are accompanied by, a disorder or inhibition of speech but are associated with a short loss of consciousness, general mental confusion and disorientation or with olfactory, gustatory, visual, or auditory phenomena (these will be discussed later). However, in my series there was no essential difference in frequency whether the lesion

2. In a few cases there was some doubt as to the type, duration and frequency of the epileptiform attacks. This may have been due to insufficient information or inadequate observation by the patient or his relatives or may have been caused by an incomplete medical investigation. The fact that the neurologist or the neurosurgeon often has no opportunity to observe the attacks may account for this.

was situated in the left or in the right hemisphere. Some of the clinical records were not detailed enough to enable one to determine definitely whether a glioma on the left side is more prone to produce aphasic signs in the attacks than a lesion on the right side. At any rate, the clinical and anatomic findings in petit mal seizures seem to justify the conclusion that they apparently have—at least in cases of glioma—relative localizing value.

3. *Jacksonian Motor Attacks.*—These were most common (50 per cent of attacks) in association with glioma in the frontoparietal area (the rolandic and postrolandic areas) and in the parietal lobe (43 per cent of attacks); they were less frequent in association with glioma in the frontal lobe (33 per cent of attacks), but they also occurred in instances of glioma in the temporal lobe (16 per cent of attacks; table 1). Therefore, these figures suggest that a diagnosis as to the location based on jacksonian motor attacks should be made cautiously. Only in cases in which there are typical attacks of constant character is it reasonably certain that the lesion is in or near the motor area; the occasional appearance of motor attacks combined with several other different types of seizures suggests a lesion in the temporal lobe.

The most frequent type of jacksonian motor attack shows the initial convulsive movement in the face, hand or distal parts of the arm. Motor attacks confined to the shoulder girdle or leg muscles are less common and indicate the presence of a tumor in the parasagittal area.

A tumor in the prerolandic parasagittal area is more likely to cause a special type of jacksonian motor attack, the so-called adversive seizure (Foerster), consisting of complex associated movements, namely, of a combined rotation of the eyes, head and neck and sometimes of the whole body. Such seizures occur mostly in association with a lesion in the upper frontal region (area 6$a\beta$ of Vogt) or, more rarely, in association with a tumor in the parietal or temporal region. Initial masticatory, phonating, grunting or swallowing movements point to a lesion near the sylvian fissure (operculum centrale). If right-handed persons suffer from motor attacks on the right side, a more or less marked inhibition of motor speech may be observed, especially in cases in which the attack starts from the facial, masticatory, phonating or deglutitory center. Attacks consisting only of transient aphasia, suggesting petit mal, have been reported. At times it is difficult to determine whether such a paroxysmal disturbance of speech without other symptoms should be considered as an epileptic equivalent.

In my material there was no patient showing isolated clonic ocular movements, which originate in the frontal eye area, area 8, according to Foerster and Penfield.

4. *Jacksonian Sensory Attacks.*—These attacks (cutaneous sensation) had a low incidence in cases of glioma purely of the frontal lobe

(4 per cent of attacks) and of the temporal lobe (6 per cent of attacks); they became more frequent in association with a lesion near the rolandic fissure (in cases of a tumor of the frontoparietal region, 19 per cent of attacks) and attained their maximum incidence in association with a tumor of the posterior part of the brain (in cases of a tumor of the parietal region, 33 per cent of attacks and in cases of a tumor of the occipital region, 33 per cent of attacks). The sensory attacks are similar to the motor attacks in that they may also be combined with inhibition of speech, verbal confusion and occasionally even with temporary word deafness.

5. *Peculiar Sensations of Abnormal Smell, Taste, Vision or Audition.*—These phenomena, preceding an epileptic seizure, are usually designated as sensory auras. From the diagnostic point of view, however, these perverted sensations should not be regarded as mere forerunners of a convulsion; in my opinion, they are the most essential part of the whole attack, since they apparently indicate the site of the primary discharge. In many cases they may give the only localizing clue. In other words, a sensory aura is possibly analogous to the primary spasmodic contraction in a jacksonian motor attack. The following motor discharge (convulsion), regardless of its character, reveals nothing more than generalization or spread of the seizure. Paroxysms of abnormal sensations representing irritative phenomena of sensory organs which are not combined with a typical epileptic attack are generally described by the term hallucination. It is highly probable that a great number of such hallucinations represent the equivalent of a localized epileptic attack in which the factor of generalization or spread is absent; at any rate, this point of view certainly holds true for patients who have not only pure hallucinations but also a combination of the same type of hallucinations with convulsions.[3]

(*A*) Paroxysmal Disturbances of Smell and Taste (So-Called Uncinate Attacks): These were most commonly encountered in association with glioma of the temporal lobe; they represented 18 per cent of attacks observed in cases of glioma of the temporal area, 18 per cent in cases of glioma of the temporo-occipital area, 7 per cent in cases of glioma of the temporofrontal area and 14 per cent in cases of glioma of the temporoparietal area. They were also frequent in association with glioma of the parieto-occipital area (14 per cent of attacks). These attacks may be accompanied by some clouding of consciousness or a petit mal attack or may be followed by a generalized convulsion. They may also be associated with sniffing, smacking movements of the lips or grunting or with a peculiar choking sensation in the throat. It seems

3. I am not including here the hallucinations observed in many cases of purely psychiatric conditions.

to be well established that uncinate attacks are due to a lesion of the secondary and tertiary olfactory and gustatory neurons in the hippocampus. This anatomic fact explains their frequency in cases of glioma of the basal part of the temporal area or of the temporo-occipital area.

(*B*) Visual Irritative Phenomena: These sensations initiating a convulsion or constituting mere equivalents in the previously mentioned sense are extremely manifold. As might be surmised, they occurred chiefly in association with a glioma near or within the visual pathways: in 37 per cent of seizures observed in the cases of glioma of the temporo-occipital region, in 33 per cent of seizures observed in the cases of glioma of the occipital region, in 7 per cent of seizures observed in the cases of glioma of the temporal region and in 7 per cent of seizures observed in the cases of glioma of the temporofrontal region. The nearer the lesion is situated to the calcarine area, the more commonly the visual attacks take the character of unformed, primitive photomas (flashes of light, scintillating colors), frequently of hemianopic type. In cases of a more anteriorly placed lesion (one in the temporal lobe), the visual phenomena are of a complex nature, such as formed visual hallucinations, which may reappear in a stereotyped manner, micropsia, macropsia, images distorted in a peculiar way (metamorphosia), "kalopsia," déjà vu and dreamy states. The last two are peculiar mental states in which the optic phenomena represent only the basis for a complicated psychologic process taking place at a higher level (Penfield and Gage, van Bogaert, Horrax, Hauptmann, Hartmann and others).

(*C*) True Acoustic Auras or Auditory Hallucinations: These are rare in cases of glioma and were noted in only one instance, in association with glioma of the temporo-occipital region. It is doubtful whether spells of "light-headedness" and dizziness observed in cases of tumor of the temporal lobe should be regarded as epileptic equivalents. Some experiences suggest that possibly they are discharges of the cortical vestibular areas (E. A. Spiegel, Stauder).

6. *Atypical Attacks.*—These may be connected with ordinary grand mal attacks as an aura, although they may also occur independently. Most of these phenomena belong to the vegetative sphere. They consist of precordial pain, palpitation, epigastric pain, belching and retching, "goose-pimple" eruption and shivering and blushing spells. As such attacks are ambiguous in regard to the diagnostic and pathophysiologic interpretation, little attention has been paid to their possible localizing value, but it is my impression that the goose-pimples and shivering attacks are not uncommon in cases of glioma of the temporal region (Stone reported 2 cases that he observed personally). They are possibly due to an irritation of the neighboring hypothalamic region. The recent investigations of J. F. Fulton and his collaborators suggest

that lesions of the premotor areas also may be responsible for vasomotor signs in epileptic attacks.

From the data cited, some conclusions can be formed as to the localizing value of attacks in cases of cerebral glioma. If three or more entirely different types of attack are present, it is probable that the tumor is located in the temporal or temporo-occipital lobe. This polymorphism of seizures is not surprising, since tumors of the temporal lobe occupy the middle of the cerebrum and may encroach on more pathways than do neoplasms of any other area. The attacks observed in cases of a lesion of the temporal lobe consist of (1) generalized major seizures and (2) petit mal attacks, without or in combination with (3) uncinate attacks (gustatory or olfactory), (4) dreamy states, déjà vu or other types of complex visual hallucinations, (5) occasionally jacksonian motor or sensory attacks and (6) vegetative phenomena, such as goose-pimples and shivering attacks.

In cases of glioma of the frontal area there is a much more uniform picture: (1) generalized grand mal attacks; (2) petit mal attacks, and (3) if the lesion is near the central fissure, jacksonian motor fits.

Glioma in the rolandic or parietal area is accompanied by motor or sensory attacks or by both, but there is only a slight tendency to generalized attacks without localizing signs. Tumor of the occipital region is characterized by a higher percentage of generalized convulsions and sometimes by primitive photomas, which may be an epileptic equivalent. There are jacksonian sensory attacks if the glioma grows toward the parietal region and uncinate or complex visual phenomena when it invades the temporal lobe.

RELATIONSHIP BETWEEN EPILEPTIC ATTACKS AND THE HISTOLOGIC TYPE OF THE GLIOMA

I have used the classification of Bailey and Cushing because it is now generally accepted. Only those cases in which the tumor could be clearly classified have been selected. It must be kept in mind, however, that a small specimen removed by operation may not reveal the true character of a glioma; the structure of the tumor often varies greatly in its different parts. Examination of a surgically removed fragment may suggest the diagnosis of a protoplasmic astrocytoma in a case in which the tumor proves to be a glioblastoma at autopsy. The material utilized for this paper has been reported on by different authors, and there was sometimes an individual difference of opinion as to how to classify a certain tumor (personal equation).

When the different subgroups of glioma are compared, it becomes obvious that the oligodendroglioma was associated with the highest

incidence of epileptic attacks, namely, in 25 of 33 cases (table 2). In other words, the ratio of the number of cases in which epilepsy occurred to the number in which it did not occur is 3.1 : 1,[4] corresponding to a percentage of 75.8. Astrocytoma follows, with an epilepsy index of 1.9 (65.5 per cent). Astrocytoma representing transitional forms to oligodendroglioma caused epilepsy in all instances (100 per cent). In the cases of the rare astroblastomas there was a slightly lower frequency of epileptic attacks, with an index of 1.4 (58.3 per cent). In the cases of glioblastoma there was a further considerable decrease in seizures, with an index of only 0.64 (39.2 per cent). The lowest index was attained by the ependymoma. In this group epilepsy occurred in only 23.5 per cent of the cases, which corresponds to an index of 0.33. It is remarkable that the frequency of epilepsy runs practically parallel with the slowness and benignity of the growth

TABLE 2.—*Frequency of Epilepsy and Calcification in Relation to Type of Glioma*

Type of Glioma	Number	With Epilepsy	Without Epilepsy	Epilepsy Index	Epilepsy in All Cases, per Cent	Calcification: Number of Cases	Calcification: Per Cent	Autopsy
Oligodendroglioma	33	25	8	3.10	75.8	25	75.8	6
Astrocytoma	113	74	39	1.90	65.5	15	13.4	38
Fibrillary	56	36	20	1.80	64.3	7	12.5	
Protoplasmic	29	16	13	1.20	55.2	5	17.2	
Astrocytoma plus oligodendroglioma	3	3	..	....	100.0	2	66.6	
Astroblastoma	12	7	5	1.40	58.3	..	....	8
Glioblastoma	125	49	76	0.64	39.2	1	0.8	66
Ependymoma	17	4	13	0.33	23.5	9	52.0	1

of the tumor (with the exception of ependymoma). In the face of these clear differences between the various types of glioma, the objection could be raised that they are due only to the possibility that the various types of glioma prefer certain areas of the brain. It was, therefore, a next step to discover whether such a preferential location of glioma exists.

Irrespective of the histologic character, glioma often seems to develop at the following locations: (*a*) the angle formed by the median surface of the cerebrum with the convexity at the longitudinal fissure; (*b*) the angle between the median surface and the corpus callosum; (*c*) the depth of the sylvian fissure; (*d*) the hippocampus and the fornix (hippocampic fissure). Figures 1 and 2 illustrate schematically the preferential location of the different gliomas, their greater or lesser frequency being indicated by a darker or lighter shading. The exact figures are given in table 3. As the frontal lobe is the largest part of

4. The ratio of the number of cases in which epilepsy occurred to the number in which it did not occur will be referred to in this paper as the epilepsy index.

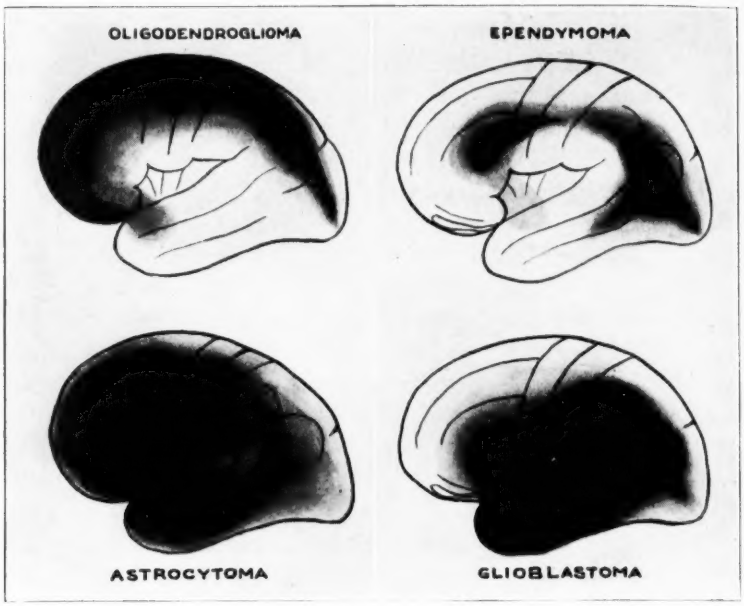

Fig. 1.—Drawings of the outer aspect of the hemisphere, showing the preferential location of the different gliomas. In this figure and in figure 2 greater or lesser frequency is indicated by darker or lighter shading.

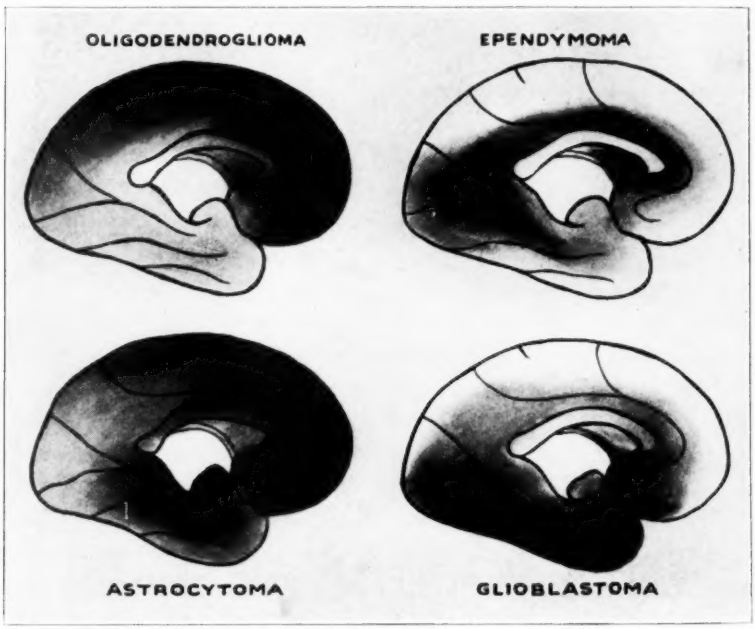

Fig. 2.—Drawing of the inner aspect of the hemisphere, showing the preferential location of the different gliomas.

TABLE 3.—*Incidence of the Different Types of Glioma in Different Cerebral Areas*

Region	Total Number	Oligodendroglioma		Astrocytoma		Astroblastoma		Glioblastoma		Ependymoma	
		Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent
Frontal	73	11	33.3	38	33.6	1	8.3	20	16.0	3	17.6
Frontoparietal	48	10	30.4	18	15.9	2	16.7	15	12.0	3	17.6
Frontotemporal	19	1	3.0	6	5.3	1	8.3	10	8.0	1	6.0
Temporal	65	1	3.0	25	22.2	3	25.0	36	28.8	..	....
Parietal	26	5	15.2	5	4.4	3	25.0	12	9.6	1	6.0
Parietotemporal	11	..	....	4	3.5	1	8.3	6	4.8	..	....
Frontoparietotemporal	4	..	....	..	....	..	....	4	3.2	..	....
Occipital	10	1	3.0	3	2.7	1	8.3	2	1.6	3	17.6
Temporo-occipital	15	1	3.0	5	4.4	..	....	6	4.8	3	17.6
Parieto-occipital	16	3	9.1	4	3.5	..	....	6	4.8	3	17.6
Frontoparieto-occipital	2	..	....	..	....	..	....	2	1.6	..	....
Temporoparieto-occipital	6	..	....	3	2.7	..	....	3	2.4	..	....
Whole hemisphere	3	..	....	2	1.8	..	....	1	0.8	..	....
Fornix and corpus callosum	2	..	....	..	....	..	....	2	1.6	..	....
Total	300	33	11.0	113	37.7	12	4.0	125	41.7	17	5.6

the brain and the occipital lobe the smallest, it is to be expected that all types of glioma are likely to occur more frequently in the anterior rather than in the posterior part of the brain, a fact which should be remembered when comparing the figures for the different types of tumor.

1. The oligodendroglioma is most common in the frontal lobe (in 33.3 per cent of the cases) and in the frontoparietal area (in 30.4 per cent of the cases); in other words, in nearly two thirds of the cases this tumor occurs in the anterior part of the cerebrum. In the posterior half of the brain the number decreases rapidly (this type of glioma occurs in the parietal region in 15.2 per cent of the cases, in the parieto-occipital region in 9.1 per cent and in the occipital region in 3 per cent). It is uncommon in the temporal lobe and in the region of the sylvian fissure. In the frontal and frontoparietal areas the oligodendroglioma has a definite tendency to grow parasagittally (types *a* and *b*). In the frontal lobe there is also a preference for the orbitomedian part of the lobe and the pole.

2. The astrocytoma is much more equally distributed over the brain than any other group. It is the most diffusely growing glioma. In some cases the astrocytoma shows no limit, the entire brain being involved. Nevertheless, the astrocytoma also shows some preference for the frontal area (in 33.6 per cent of the cases) and the frontoparietal area (in 15.9 per cent of the cases) and is also less common in the posterior part of the brain, which may be explained by the larger size of the frontal lobe. In contrast to the oligodendroglioma, it often (in 22.2 per cent of the cases) occurs in the temporal lobe (type *d*). It shows no marked predilection for the sylvian fissure but is not rare in the parasagittal area. Astrocytoma in the parasagittal area frequently grows through the corpus callosum into the other hemisphere (fig. 3).

3. The astroblastomas studied are too few to be evaluated, but, in contrast to the astrocytomas, they seem to favor the parietal area rather than the frontal lobe.

4. The glioblastoma shows an almost reverse distribution from the oligodendroglioma. Its usual site is the region surrounding the sylvian fissure, with a maximum frequency in the temporal lobe (type *c* or *d*). In 52 per cent of all the cases of glioblastoma the temporal lobe was somehow involved; in 28.8 per cent the lesion was limited solely to the temporal lobe. There is a definite predisposition for the posterior half of the cerebrum, with involvement at the same time of the temporal, parietal and occipital lobes. Glioblastoma of the frontal lobe occurs usually near the sylvian fissure, in or beneath the third frontal convolution. It occurs less commonly on the median surface of the lobe and rarely at the pole.

5. An analysis of the group of ependymomas reveals that the distribution corresponds roughly to the outline of the cerebral ventricles. Although this tumor arises from ependymal cells, it must be emphasized that this neoplasm is not confined to the subcortical areas; in my series it reached the surface of the brain in the majority of cases. There was a striking preference for the junction of the parieto-occipital and temporo-occipital areas, almost one half of all ependymomas being situated there. It is highly suggestive that this location corresponds to the ventricular triangle, where the temporal and occipital horns meet the midportion of the lateral ventricle. Another center of growth for the cerebral ependymomas lies more anteriorly in the frontal or frontoparietal area approximately corresponding to the anterior horn and its junction with the third ventricle (foramen of Monro).

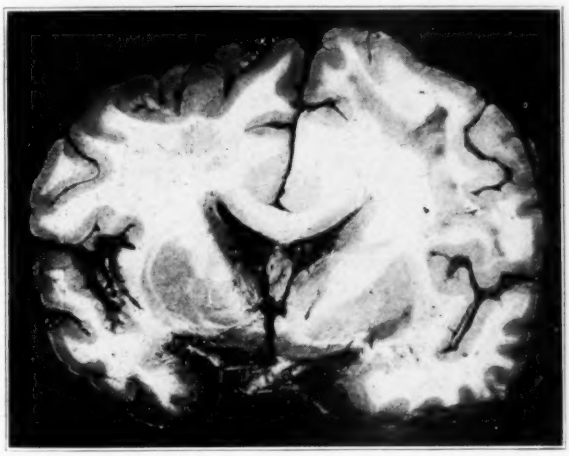

Fig. 3.—Cross-section of a diffuse astrocytoma of the left frontoparietal area. The infiltrated gray matter is poorly outlined. There is marked swelling of the white matter. The tumor infiltration spreads through the widened corpus callosum to the gyrus cinguli of the right side.

After this tentative analysis of the preferential site of the different gliomas, one must admit that some of the differences in the incidence of epilepsy are undoubtedly due to such variations in location. However, when the incidence of epilepsy in association with tumors of different histologic type but in the same gross location is compared, it can be definitely proved that the histologic character of the lesion is another important factor for the production of epileptic attacks (table 4).

Among a total number of 73 gliomas limited to the frontal lobe, there were 11 oligodendrogliomas, 63.6 per cent of which were associated with epilepsy; 38 astrocytomas, 50 per cent of which were associated with epilepsy; 20 glioblastomas, 55 per cent of which were

TABLE 4.—*Incidence of Epilepsy in Cases of Glioma in the Same Location but of Different Histologic Type*

Region	Total Number	Oligodendroglioma			Astrocytoma			Astroblastoma			Glioblastoma			Ependymoma		
		With Epilepsy	Without Epilepsy	Percentage of All Cases with Epilepsy	With Epilepsy	Without Epilepsy	Percentage of All Cases with Epilepsy	With Epilepsy	Without Epilepsy	Percentage of All Cases with Epilepsy	With Epilepsy	Without Epilepsy	Percentage of All Cases with Epilepsy	With Epilepsy	Without Epilepsy	Percentage of All Cases with Epilepsy
Frontal	73	7	4	63.6	19	19	50.0	1	..	100.0	11	9	55.0	..	3	0.0
Frontoparietal	48	10	..	100.0	17	1	94.5	2	..	100.0	9	6	60.0	..	3	0.0
Frontotemporal	19	1	..	100.0	6	..	100.0	..	1	0.0	1	9	10.0	..	1	0.0
Temporal	65	1	..	100.0	17	8	68.0	1	2	33.3	14	22	38.8	..	..	....
Parietal	26	3	2	60.0	4	1	80.0	2	1	66.6	4	8	33.3	..	1	0.0
Parietotemporal	11	..	..	....	2	2	50.0	1	..	100.0	3	3	50.0	..	..	....
Frontoparietotemporal	4	..	..	....	..	..	....	..	..	....	3	1	75.0	..	..	....
Occipital	10	..	1	0.0	1	2	33.3	..	1	0.0	..	2	0.0	1	2	33.3
Temporo-occipital	15	1	..	100.0	3	2	60.0	..	..	....	1	5	16.6	1	2	33.3
Parieto-occipital	16	2	1	66.6	1	3	25.0	..	..	....	1	5	16.6	2	1	66.6
Frontoparieto-occipital	2	..	..	....	..	..	....	..	..	....	1	1	50.0	..	..	....
Temporoparieto-occipital	6	..	..	....	2	1	66.6	..	..	....	..	3	0.0	..	..	....
Whole hemisphere	3	..	..	....	2	0	100.0	..	..	....	..	1	0.0	..	..	....
Fornix and corpus callosum	2	..	..	....	..	..	....	..	..	....	1	1	50.0	..	..	....

associated with epilepsy, and 3 ependymomas not associated with epilepsy. (The astroblastomas have been discarded on account of their scarcity.) Among the group in the frontoparietal (rolandic and postrolandic) area, 100 per cent of the oligodendrogliomas and 94.5 per cent of the astrocytomas were associated with epilepsy, but only 60 per cent of the glioblastomas. None of the 3 ependymomas was associated with epilepsy. Among 19 tumors of the frontotemporal region, all the astrocytomas present were associated with epilepsy, but only 10 per cent of the glioblastomas caused attacks. The gliomas in the temporal region revealed the same tendency: One oligodendroglioma was associated with epilepsy, while 68 per cent of the astrocytomas and 38.8 per cent of the glioblastomas led to attacks. In cases of a lesion of the parietal region, epileptiform attacks occurred in association with 60 per cent of the oligodendrogliomas, 80 per cent of the astrocytomas, 33.3 per cent of the glioblastomas and none of the ependymomas. In cases of glioma of the occipital, temporo-occipital and parieto-occipital regions the highest incidence of epilepsy again occurred with oligodendroglioma; astrocytoma followed, and the lowest percentage occurred in the cases of glioblastoma. Within this group, however, the ependymoma showed an elevated figure for attacks, averaging about the same level as that for the astrocytoma.

From the foregoing tabulation it may be concluded that, regardless of location, oligodendroglioma predominates in the production of epilepsy and astrocytoma is second, whereas glioblastoma ranks considerably lower. Ependymoma falls somewhat out of line, as it produces a low incidence of epilepsy in locations where other types of tumor cause a high one. However, this tumor shows a surprisingly high epilepsy index when it is in the occipital, parieto-occipital or temporo-occipital region. It is possible that the relatively small number of ependymomas does not rule out the errors caused by statistical hazard.

These striking differences having been established, the type of epileptic attacks associated with the major types of glioma in the same gross location (table 5) may be studied.

When oligodendroglioma, astrocytoma and glioblastoma in the frontal region are compared, it becomes evident that in cases of oligodendroglioma and, to a lesser degree, those of astrocytoma, there is a very high percentage of generalized convulsions without localizing signs and also of petit mal attacks. However, there is a relatively low percentage of focal attacks of the jacksonian motor type. In cases of glioblastoma in the frontal region there is a relatively high incidence of epilepsy in general (table 4) and especially of the jacksonian motor type (table 5). This is probably to be explained by the fact that most of these tumors lie in close proximity to the lower end of the precentral

TABLE 5.—*Comparison of the Type of Attacks Observed in Cases of the Major Types of Glioma in the Same Location*

Region	Total Number	Type of Epilepsy: General		Petit Mal		Jacksonian Motor Attacks		Jacksonian Sensory Attacks		Uncinate Attacks		Visual Attacks	
		Number	Per Cent*	Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent
Frontal													
Astrocytoma	19	14	50	3	11	6	22	2	8	2	8	..	..
Glioblastoma	11	4	31	..	..	9	69	..	..	..	..	..	..
Oligodendroglioma	7	5	63	2	25	1	12	..	..	..	..	..	..
Temporal													
Astrocytoma	17	12	41	6	21	2	7	2	7	4	14	3	10
Glioblastoma	14	5	27	1	6	6	33	1	6	4	22	1	6
Frontoparietal													
Astrocytoma	17	5	21	1	4	15	63	3	12	..	..	..	..
Glioblastoma	9	1	8	2	17	4	33	4	33	1	8	..	..
Oligodendroglioma	10	4	29	2	14	6	43	2	14	..	..	..	..
Parietal													
Astrocytoma	4	1	20	..	..	2	40	2	40	..	..	..	..
Glioblastoma	4	3	42	..	..	2	29	2	29	..	..	..	..
Oligodendroglioma	3	..	..	..	..	2	50	2	50	..	..	..	..

* The percentages refer to the total number of attacks.

gyrus and to the sylvian vessels. The latter point will be further commented on. A similar difference exists between astrocytoma and glioblastoma in the temporal region. In cases of tumor in the frontoparietal region the incidence of generalized epilepsy is again low for glioblastoma but rather high for astrocytoma and oligodendroglioma. The jacksonian motor attacks are also much more frequent in association with the two latter types, whereas the sensory attacks appear to be more common in the cases of glioblastoma in this location. In cases of lesions in the parietal region the proportion is reversed, as here the astrocytomas and oligodendrogliomas outnumber the glioblastomas in regard to the incidence of jacksonian motor as well as jacksonian sensory attacks, but there is a greater tendency for generalized seizures in cases of glioblastoma.

GROSS AND MICROSCOPIC ANATOMIC CHANGES AND THEIR IMPORTANCE IN THE DEVELOPMENT OF EPILEPTIC ATTACKS

As has already been stated, neoplasms in the same location but of different histologic types vary much in the percentage of epileptiform attacks. This must be explained chiefly on histologic grounds. It is realized that the problem of epilepsy is not merely anatomic and that neurophysiologic factors must also be considered. According to an accepted conception of experimental neurophysiology, epileptic attacks have been regarded as manifestations of neuronal irritation. Hence the question arises whether attacks occur more often in cases of cortical tumor than in cases of subcortical tumor. Careful macroscopic and microscopic study of autopsy specimens revealed that few gliomas were confined solely to the cortex or to the subcortical white matter. It cannot be denied, however, that glioblastoma and especially ependymoma showed a more marked tendency to grow subcortically than the other groups, and in these cases there was also a lower incidence of epileptic attacks. In spite of this fact, there were so many exceptions to the rule that it was not considered justifiable to draw any definite conclusion in an individual case as to the significance of the cortical or subcortical location of a glioma as a factor of importance in causing the epilepsy.

1. *Meningeal Adhesions or Spreading of the Tumor Within the Meninges.*—These occur only occasionally in cases of glioma, but in my observations the tendency for epilepsy to develop was no more marked in such cases than in cases without meningeal involvement.

2. *Size of the Tumor in Relation to Epileptiform Attacks.*—It might be assumed that very large tumors cause more frequent attacks, as they encroach on a greater number of pathways or cortical areas. On the

other hand, a small tumor may cause attacks (fig. 4), and occasionally a very large tumor, infiltrating almost the whole brain, fails to produce them (fig. 5). Glioblastoma may cause a few attacks in its initial stages, which disappear later with the expansion of the neoplasm.

3. *Manner of Growth.*—An important point in the problem is the manner in which a glioma grows, whether it is diffusely infiltrating or sharply demarcated or merely displaces the surrounding brain substance. It is my impression that epileptic attacks occur most fre-

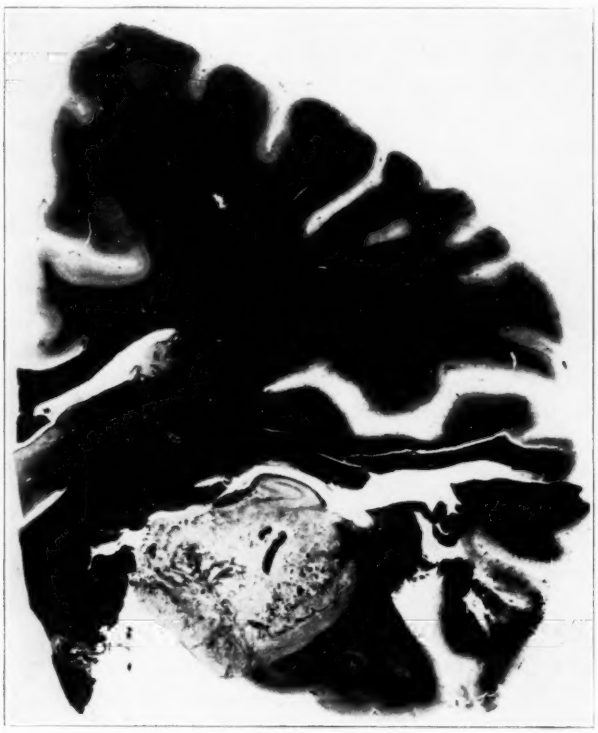

Fig. 4.—Small glioblastoma of the left gyrus hippocampi (Weigert's myelin stain). The tumor is sharply demarcated and very necrotic, with large thrombosed vessels. There is only slight demyelinization of the surrounding white matter. Clinically, there were generalized epileptic attacks.

quently in cases of diffusely infiltrating glioma (fig. 3) in which many ganglion cells and nerve fibers remain fairly intact between the growing tumor cells. Thus, the main effect of the tumor may be an irritating influence on the neurons manifesting itself in epilepsy but not leading to complete anatomic and functional destruction. Such a condition is typical for many cerebral astrocytomas and is also frequent in cases of oligodendroglioma. A large number of astrocytomas are of extremely

slow growth, the cells being loosely scattered over a large area without doing much damage to the enclosed nerve elements (fig. 6). It is remarkable that they show comparatively little vascular involvement and subsequent regressive changes. In oligodendroglioma there is usually a widespread infiltrating proliferation of oligodendroglia cells in the brain tisue surrounding the grossly visible tumor mass. These anatomic peculiarities may account for the frequency of irritative symptoms as well as the absence or paucity of neurologic deficiency symptoms [5] in cases of astrocytoma and oligodendroglioma. The growth of glioblastoma is different; it not infrequently has a well defined line of demarcation (at least macroscopically). It forms a solid tumor mass which usually undergoes early and extensive degeneration and necrosis, owing

Fig. 5.—Astrocytoma of the left hemisphere (Weigert's myelin stain), with diffuse neoplastic infiltration of the brain and severe diffuse demyelinization. Clinically, there were no epileptic attacks.

to severe vascular changes, and therefore it contains scarcely any intact nerve cells or fibers. The described differences between glioblastoma and astrocytoma are clearly demonstrated by the Weigert myelin stain. Whereas astrocytoma shows a wide area of diffuse, poorly delimited, usually incomplete demyelinization, glioblastoma is more likely to produce complete loss of myelin in a well demarcated area (figs. 4, 5 and 7). Hence, it is not astonishing that glioblastoma clinically causes more deficiency symptoms than irritative symptoms. In contrast to these forms of glioma, ependymoma often grows in a well demarcated manner, dis-

5. Deficiency symptoms are those due to loss of function dependent on the destruction of nerve tissue.

Fig. 6.—Motor area of the same brain as in figure 3 (Nissl's stain). Well preserved ganglion cells are present within the gliomatous area.

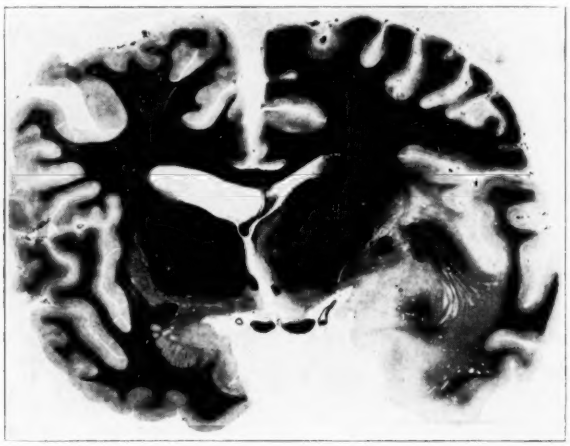

Fig. 7.—Astrocytoma of the right temporal lobe (Weigert's myeliu stain), with diffuse and poorly demarcated demyelinization. Clinically, there were generalized epileptic attacks, with an aura of fulness in the throat and nausea.

placing the adjacent structures. As its expansion is usually directed from the ventricle toward the cortex, it seems to cause relatively little deficiency or irritative phenomena.

4. *Richness of Glia Fibrils.*—There is little doubt that the higher incidence of epilepsy parallels the richness of glia fibrils. If one compares, for example, fibrillary and protoplasmic astrocytoma in the same location, a higher percentage of epilepsy occurs in association with the fibrillary type (table 6). The epilepsy index for fibrillary astrocytoma is 1.8, and that for protoplasmic astrocytoma is 1.2 (table 2). B. Schlesinger reached similar conclusions in his paper. The presence of glia fibrils is believed to have a mechanically irritating effect, which increases the preparedness for attacks, a fact well established by observations of the glial mesenchymal scars produced by cerebral injuries (Penfield and Foerster).

TABLE 6.—*Frequency of Epileptic Attacks in Cases of Fibrillary and Protoplasmic Astrocytoma*

	Fibrillary Astrocytoma				Protoplasmic Astrocytoma			
Region	With Epilepsy	Without Epilepsy	Percentage of Cases with Epilepsy	Epilepsy Index	With Epilepsy	Without Epilepsy	Percentage of Cases with Epilepsy	Epilepsy Index
Frontal.....	9	11	45.0	0.80	3	5	37.0	0.6
Temporal.....	8	4	66.7	2.00	3	3	50.0	1.0
Frontotemporal.	2	0	100.0	....	3	0	100.0	...
Frontoparietal..	10	0	100.0	....	2	1	66.7	2.0
Parietal.....	..	..	....	....	2	1	66.7	2.0
Parieto-occipital	2	3	40.0	0.67	..	..	....	....
Temporo-occipital	2	..	100.0	....	1	2	33.3	0.5

5. *The Presence of Cysts.*—This feature seems to play no rôle in the production of epilepsy. Among 74 astrocytomas associated with epilepsy, 17 (23 per cent) were cystic; among 39 astrocytomas not associated with epilepsy, 9 (23.1 per cent) were cystic.

6. *Calcification.*[6]—This is an interesting and important factor in the production of attacks. If one excludes ependymoma, there was apparently a perfect parallelism between the frequency of epilepsy and that of calcification (table 2). In the cases of oligodendroglioma, for example, with its high epilepsy index, there was also the highest percentage of calcification (75.8), whereas in the cases of glioblastoma the figure was very low for both. The deposits of calcium may act in the same mechanically irritative way as do the glia fibrils. In cases of oligodendroglioma and in some of astrocytoma calcification is present not only in the central parts of the tumor but also at the borderline of the neoplasm or even in the adjacent brain tissue (van Dessel). Ependy-

6. Only calcifications visible in the roentgenograms are considered here.

moma was associated with the lowest incidence of epilepsy, and yet it often contained calcifications (in 52 per cent of the cases). No satisfactory explanation can be given for such behavior, but it may be conjectured that the calcification in an ependymoma does not act as an irritant on the nerve elements, since it is located chiefly in the center of a solid tumor mass.

7. *The Vascular Factor.*—In recent years much attention has been paid to the vascular factor in epilepsy (Kennedy, Foerster, Sargent, MacRobert and Feinier and Penfield). It must be admitted that with this point of view there is a departure from the purely anatomic ground, and the problem is put on a wider and more complex physiologic basis. It has been shown that the major epileptic attack is preceded by a spasm of the cerebral arteries (Foerster) and that spasms of cerebral arteries or localized cerebral anemia may cause an epileptic seizure.

In cases of intracranial tumor, undoubtedly, conditions often exist which favor the occurrence of such spasms or of vascular compression leading to more or less transitory local anemia. It is the opinion of MacRobert and Feinier that the epileptic attacks in cases of glioma of the temporal region are due to impairment of circulation in the sylvian vessels and their branches (that is, produced by direct pressure or neighboring edema). Although absolute anatomic proof is lacking, this theory sounds feasible. There were instances in Dr. Lowenberg's collection of myelin sections through the whole brain which showed clearly the intimate relationship of tumors in the basal part of the temporal region to the middle cerebral artery and its branches (figs. 4 and 7). A temporary interference with the cortical blood supply would explain well the fact that in cases of glioblastoma of the frontal and temporal regions there is such a high percentage of jacksonian motor attacks in comparison with other groups of glioma. Most of the glioblastomas of the frontal and temporal regions lie near enough to the anterior parts of the sylvian fissure to interfere with the blood supply of the posterior frontal and precentral areas, which are supplied by the truncus ascendens: arteria orbitofrontalis, arteria praecentralis and arteria rolandica (nomenclature of Foix). The frequency of jacksonian sensory attacks in cases of glioblastoma of the frontoparietal region may be interpreted in the same way. In these cases the tumor surrounds the middle or posterior part of the sylvian fissure and disturbs the circulation in the postrolandic and parietal region (arteria parietalis anterior and arteria parietalis posterior).

On the other hand, the rarity of epileptic attacks in cases of ependymoma is due possibly to the fact that this tumor develops from inside to outside and causes little and late, if any, interference with the cortical blood supply. The question of calcification, too, seems to be indirectly

connected with that of the circulation of the blood, for the presence of deposits of calcium always reveals nutritional damage caused by local anemia and the effect of abnormal metabolic products. I have already mentioned that calcifications are not confined to the necrotic central parts of the tumor but occur also in the viable peripheral parts of the neoplasm, where the walls of small hyalinized vessels are the seat of deposits of calcium. It is common for such vascular alterations to spread widely beyond the limits of the tumor into otherwise well preserved brain tissues. While it is possible that the deposits of calcium may cause some mechanical irritation, it is more probable that both the epileptic attacks and the calcification represent a secondary manifestation of primary vascular damage in the surrounding cortex.

DIFFERENTIAL DIAGNOSIS AND PROGNOSIS

The anatomic changes potentially responsible for the occurrence of epileptic attacks having been analyzed, it seems worth while to bring

TABLE 7.—*Duration of Epilepsy in Patients with Different Types of Glioma Prior to Admission*

	0 to 3 Months	3 Mos. to 1 Year	1 to 2 Years	2 to 5 Years	5 to 10 Years	More Than 10 Years
Oligodendroglioma	....	14%	18%	31%	23%	14%
Astrocytoma	12%	32%	19%	21%	7%	9%
Astroblastoma	43%	14%	14%	29%	....	....
Glioblastoma	53%	13%	21%	13%	....	....
Ependymoma	....	67%	33%	....	....	....

out some points of value in regard to the clinical differential diagnosis and the prognosis.

In the majority of cases of cerebral glioma, epileptic seizures are an early, if not the first, sign in the chronology of the disease. Judging from the duration of epilepsy alone, it is already clinically possible to rule out certain types of glioma (table 7). If the epileptic attacks have been present for more than two years, a glioblastoma is not likely to be found; if the duration of the attacks exceeds five years, it can be definitely excluded. On the other hand, an oligodendroglioma is improbable and a glioblastoma may be suspected if epilepsy occurs during only a short episode in the disease (of less than three months' duration). The following diagnostic rules may be given for the differentiation of the various types of glioma:

1. The diagnosis of oligodendroglioma or astrocytoma must be favored if: (*a*) epilepsy appears for the first time in an adult from 20 to 40 years of age; (*b*) the attacks are of a focal type (for exceptions see p. 337), and (*c*) the attacks are the first and only symptom

for a long time, frequently for many years.[7] Both types of tumor are characterized by the frequent absence or by the late onset of symptoms of increased intracranial pressure and neurologic deficiency. These features were particularly conspicuous for oligodendroglioma, 23 per cent of the patients with this type of tumor having had epilepsy for from five to ten years and 14 per cent for more than ten years. The majority of the oligodendrogliomas (75.8 per cent) were calcified. In the case of astrocytoma, however, the duration of symptoms is somewhat shorter; intracranial pressure phenomena are more prone to develop, but the tumor is less apt to calcify.

2. Glioblastoma more often occurs in patients over 40 years of age; they usually have a history of a very short course, with only a few epileptic attacks, rapidly followed by marked neurologic deficiency symptoms, attacks having been present for less than three months in

TABLE 8.—*Average Duration of the Disease in Patients with Glioma With and Without Epilepsy*

Type of Tumor	With Epilepsy			Without Epilepsy	
	Duration of Epilepsy, Months*	From First Symptom to Admission, Months	Total Duration, Months	From First Symptom to Admission, Months	Total Duration, Months
Oligodendroglioma	65.7	76.2	99.9	16.0	27.3
Astrocytoma	39.5	43.3	52.9	11.0	17.9
Astroblastoma	18.2	19.0	40.3	14.0	29.2
Glioblastoma	9.6	11.7	19.2	3.2	5.5
Ependymoma	7.8	12.8	19.3	5.4	17.7

* Prior to admission.

53 per cent of the cases studied. Roentgen changes are not common in this group.[8]

3. Most of the cerebral ependymomas develop in childhood or adolescence. They cause early and marked increase of intracranial pressure but relatively few clinical localizing signs. While their whole course is rather short, epileptic attacks are not frequent and appear late. The ependymoma is practically the only calcifying glioma in childhood.

It is of interest that the presence of epileptiform attacks in cases of glioma of the cerebral hemisphere is a favorable prognostic sign, so far as the probable expectation of life of the patient is concerned.

As table 8 clearly demonstrates, in cases of all types of glioma, irrespective of the histologic character, the average duration of life (the time from the first-observed symptom to admission plus the postopera-

7. The presence of a meningioma should also be considered and can be recognized, in many instances, from roentgenograms.

8. In such cases a metastatic tumor of the brain must be ruled out.

tive period of survival) has been much longer for patients with epilepsy than in those without attacks.

SUMMARY

Three hundred verified and classified cases of glioma of the cerebral hemispheres have been studied with reference to epileptic attacks. After a definition of epileptiform attacks and their equivalents, some sources of error and misinterpretation are discussed. The occurrence, frequency and character of convulsive seizures depend on two main local factors: (*a*) the site and (*b*) the histologic structure of the tumor. The different types of attacks are described with reference to their localizing value and frequency in association with lesions in certain locations. Some conclusions are also formed as to whether glioma in certain areas of the brain tends to produce given epileptic syndromes.

When the frequency of the attacks is compared, marked differences are found among the various histologic types of glioma. To eliminate the factor of location, the preferential site of the different types of glioma was first considered. Histologically different types of glioma in the same gross location show a definite difference not only in the frequency but also in the type of attacks. In cases of the slow-growing types of tumor attacks are more frequent than in those of the rapidly growing lesions.

In cases of the more diffusely growing types of glioma (astrocytoma and oligodendroglioma), with their widespread infiltration of the neighboring tissues, there is a higher incidence of epileptic attacks than in cases of the usually better demarcated glioblastoma and ependymoma.

The following anatomic and physiologic factors appear to play an important rôle in the production of attacks: Richness of glia fibrils and the presence of calcifications promote attacks (an exception is ependymoma); also encroachment on important cerebral arteries by the tumor, causing temporary localized cerebral anemia or leading to permanent nutritional damage (calcification in the surrounding brain tissue).

Whether a more deeply placed subcortical glioma is less prone to produce epileptic attacks than a superficial tumor could not be accurately determined, although it seemed probable.

The size of the glioma and the presence of cysts and meningeal adhesions have no influence in the production of epileptic attacks.

Some suggestions are given as to what type of glioma may be predicted preoperatively by considering the type and duration of the attacks.

The presence of epileptiform attacks makes the prognosis more favorable, irrespective of the type of glioma, since patients with attacks have a longer period of survival and expectation of longer life than those without seizures.

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Clinical Notes

A STAIN FOR NEUROGLIA IN FORMALDEHYDE-FIXED PARAFFIN SECTIONS

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The difficulty of obtaining fresh, properly fixed tissue has unfortunately limited the usefulness of the gold and silver impregnation methods of Ramón y Cajal [1] and del Rio-Hortega [2] for demonstration of the neuroglia cells, particularly in the study of old formaldehyde-fixed material and of brains hardened intact in formaldehyde before sectioning. The deformalization and bromination proposed by Globus [3] have given only partly satisfactory results in our hands.

The necessity for using frozen sections with most previously employed metallic staining methods has also been a distinct disadvantage at times. With many friable neoplastic tissues the difficulty of cutting satisfactorily thin and uniform sections on the freezing microtome and carrying them through many different solutions off the slides has diminished the value of the methods.

We have previously, on the advice of Bailey,[4] employed Perdrau's [5] method for silver impregnation of reticulin for paraffin sections, but this is the only metallic method which we have hitherto found successful with these sections.

The method which we describe in this paper has given uniformly good results with tissue fixed in formaldehyde for periods varying from a few days to ten years. It is used for paraffin sections of material dehydrated, embedded, cut and mounted by the method employed as a routine for all other tissues in the laboratory of surgical pathology. Although the technic of impregnation is somewhat long, it is simple and is not subject to the caprice and variability so often encountered with the usual methods, and it does not interfere greatly with the other routine duties performed by the technician.

The technic employed involves a modification of Bielschowsky's [6] method for the impregnation of nerve fibers in frozen sections with the addition of treatment with heated gold chloride.

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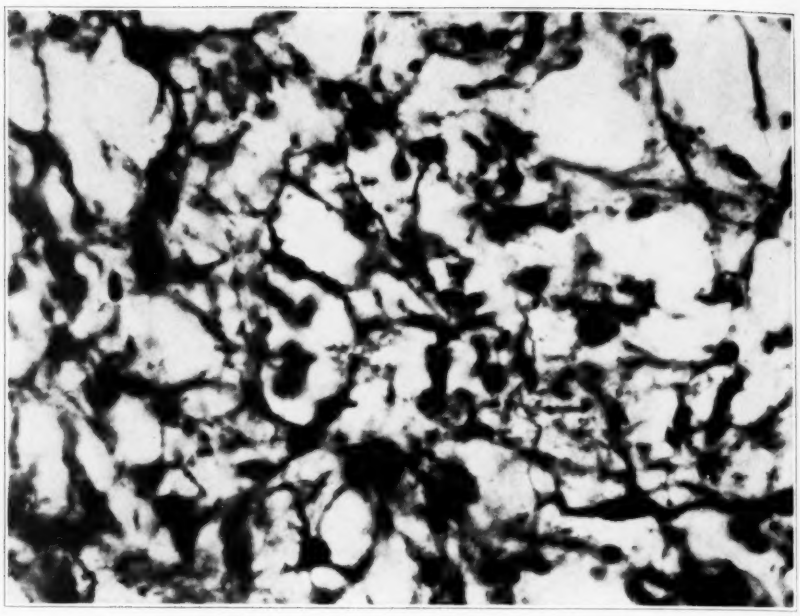

Fig. 1.—Section (×600) of an astrocytoma fixed in formaldehyde for four years. Every cell is impregnated, though all the cells are not in focus.

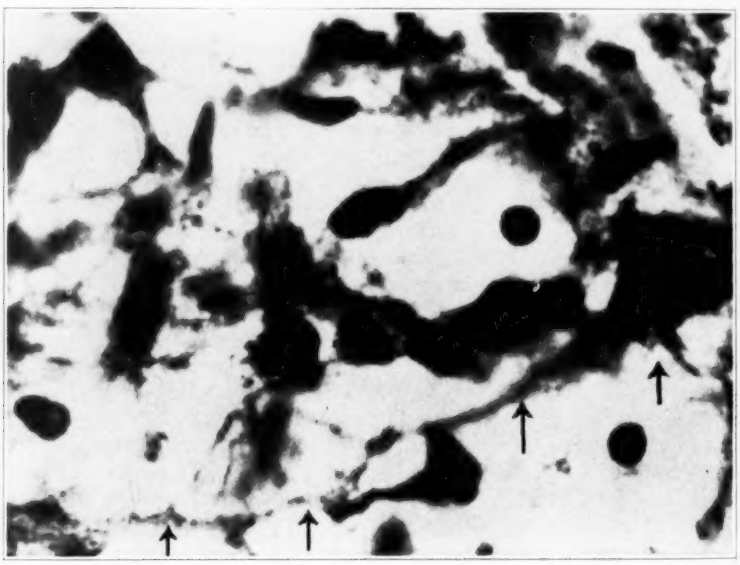

Fig. 2.—Section (×1,200) of the same tumor as that shown in figure 1. There is a large, immature astrocyte at the right of the center with a long nutritive process (indicated by arrows) and a sucker foot attached to the wall of the blood vessel at the lower left corner. In the spongioblast lying above the center astroblastic processes are beginning to develop opposite the nutritive process.

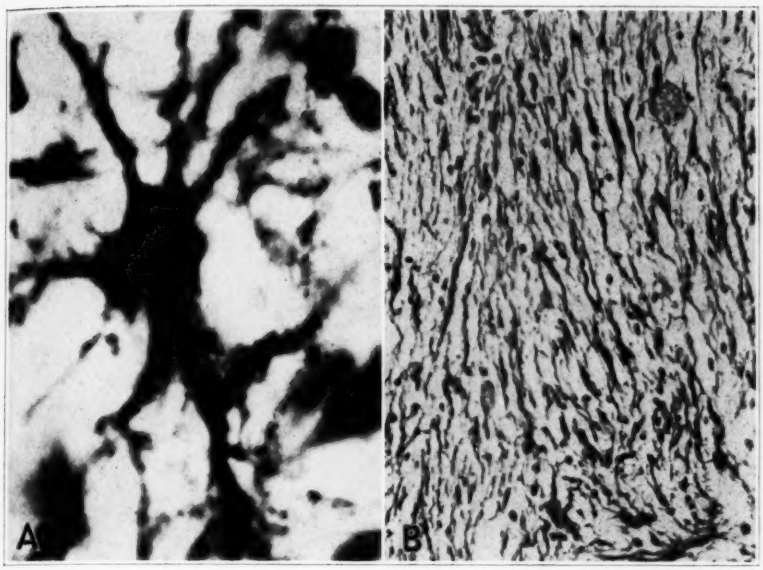

Fig. 3.—(*A*) Detail of an astrocyte from the same tumor as that shown in figures 1 and 2 (× 1,200) and (*B*) section of a spongioblastoma polare, showing the parallel arrangement of polarized spongioblasts (× 200).

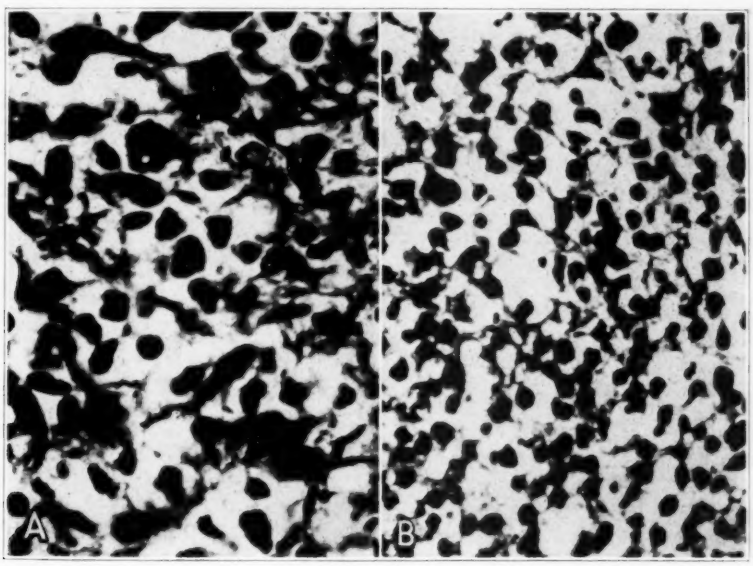

Fig. 4.—Sections of (*A*) a glioblastoma multiforme, showing the great variation in the size, shape and staining qualities of the cells (× 600), and (*B*) a medulloblastoma (× 600). The poorly developed protoplasmic extensions are better demonstrated than is usually possible in these tumors.

Since this report was begun, Loughlin[7] has published the description of a similar method involving impregnation with both silver and gold, with which he has obtained excellent results. His technic differs from that described here in four important particulars: He employs frozen sections; he passes his sections through bromine water as a preliminary step; he impregnates with gold chloride before employing Bielschowsky's silver solution, and he does not employ prolonged impregnation with silver nitrate.

Our method has been chosen after prolonged study of many variations. More than five hundred sections have been used to determine the optimum thickness of sections and the time of treatment with the various solutions. The technic which is described has given uniformly satisfactory results in the study of many

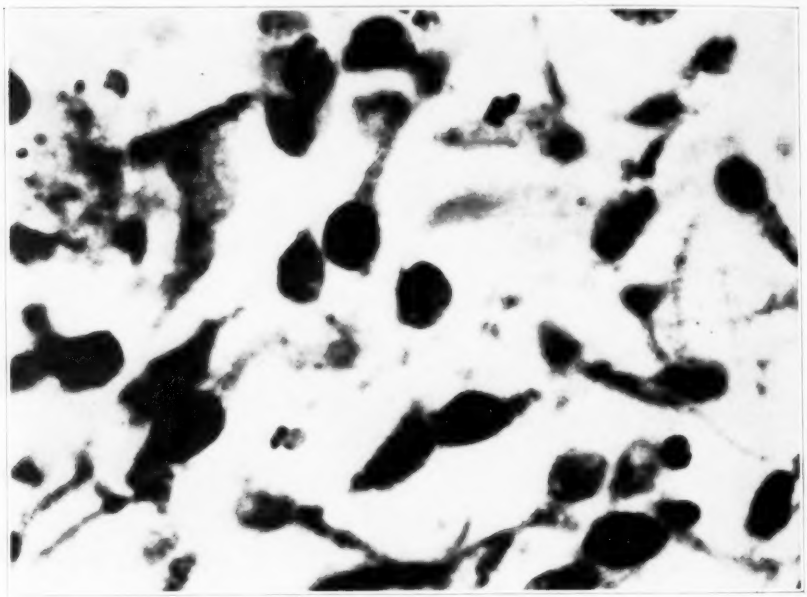

Fig. 5.—Section (× 1,200) of a transitional glioma of an unusual type.[8]

kinds of intracranial tumors and has been of value for preparations of tumors arising outside the central nervous system as well.

TECHNIC

1. Embed suitable blocks of formaldehyde-fixed tissue in paraffin according to the usual routine. (Tissue fixed in Cajal's solution of formaldehyde and ammonium bromide may be used with equally good results. Tissue fixed in solutions containing potassium bichromate is not satisfactory.)

7. Loughlin, E. H.: A Simple Routine Procedure for Staining the Neurons and Glia of the Nervous System, Arch. Neurol. & Psychiat. **33**:616 (March) 1935.

8. This tumor is reported in detail elsewhere by Dr. George S. Mahon of the Department of Pathology.

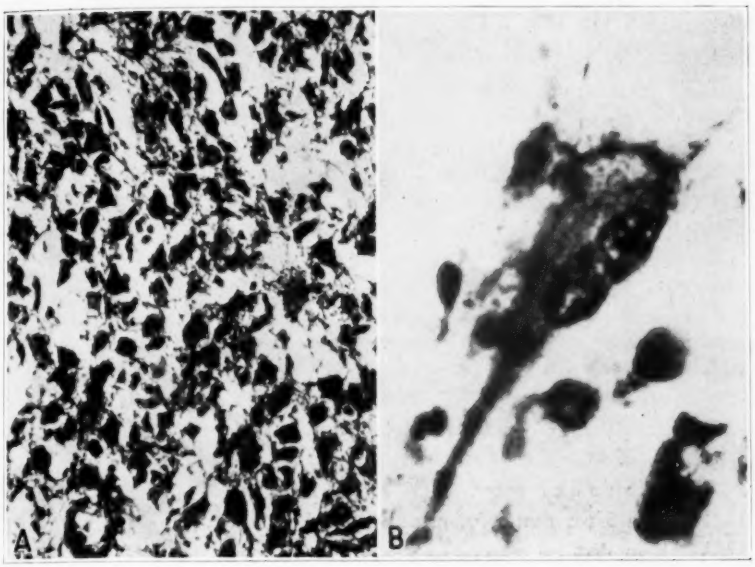

Fig. 6.—(*A*) Section of a ganglioneuroma (× 200) and (*B*) detail of a poorly developed ganglion cell from the same tumor (× 1,200).

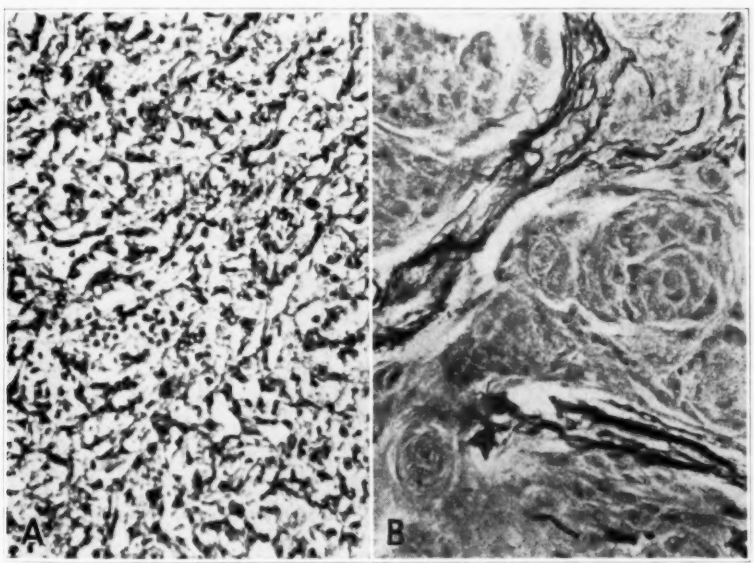

Fig. 7.—Sections (× 200) of (*A*) a cerebellar hemangioblastoma, showing impregnation of the fine reticular network and (*B*) a meningeal fibroblastoma. The framework of reticulin between the whorls of tumor cells is heavily stained, but there is no intercellular fibrillary material.

2. Cut paraffin sections at 11 microns and mount on slides with Mayer's glycerin-albumin mixture.

3. Remove the paraffin and wash in distilled water.

4. Place in a 2 per cent solution of silver nitrate in the dark for forty-eight hours.

5. Wash quickly in distilled water.

6. Place in Bielschowsky's [6] ammoniacal silver bath for two hours in the light. Bielschowsky's silver solution is made as follows: To 5 cc. of a 20 per cent solution of silver nitrate add 2 drops of a 40 per cent solution of sodium hydroxide; dissolve the precipitate with ammonium hydroxide, added drop by drop; dilute to a volume of 50 cc. with distilled water, and filter.

7. Pass slides one at a time from the silver bath quickly through two changes of distilled water into a dilute solution of formaldehyde, U.S.P. (1:10), made up with tap water.

8. Wash in two changes of distilled water.

9. Place in a bath of 1 per cent solution of yellow gold chloride in the incubator at from 50 to 60 C. for from one and a half to two hours.

10. Wash in distilled water.

11. Fix in a 5 per cent solution of sodium hyposulfite.

12. Wash in distilled water.

13. Dehydrate; clear in oil of origanum and mount in balsam.

The stain obtained by this method is by no means specific. The interstitial elements of connective tissue, the nerve cells and fibers and the neuroglia cells are impregnated equally well. The method is particularly applicable to the demonstration of the interstitial processes, not only of mature astrocytes but of the more immature cell types seen in the primitive gliomas, such as medulloblastoma and glioblastoma multiforme. Spongioblasts are invariably well impregnated. Oligodendroglia and microglia cells are demonstrated poorly in most sections. The accompanying illustrations show clearly the results that may be regularly obtained by this method for various tumors. They show also that the sections lend themselves well to photomicrography.

THE AURICULOTEMPORAL SYNDROME

With a Suggestion Regarding Therapy

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The following case of auriculotemporal syndrome is reported because of the comparative rarity of the condition and because of the interesting physiologic problem involved.

REPORT OF CASE

S. R., a white man aged 34, a machinist, entered the neurologic clinic in December 1933, complaining that as soon as he began to eat profuse perspiration appeared over the right side of the face. At about the age of 10 years he had noted a swelling in front of the right ear. When he was about 20, a surgeon operated on this mass, removing some soft tumor tissue. The operation was followed promptly by paralysis of the right side of the face. Also, very shortly after the operation, a fistulous opening appeared at the site of the operation, from which some fluid exuded whenever he ate. About six months after the first operation a physician applied some substance (a caustic?) to this opening and succeeded in effecting its closure. Thereafter, whenever the patient ate, instead of fluid flowing from the fistula, there rapidly appeared marked sweating over the right cheek. The reaction was most pronounced when he ate spicy foods.

General medical examination gave negative results. Neurologic examination disclosed paresis of the right side of the face. Associated movements, affecting the corner of the mouth, were present whenever the patient closed his eyes; taste was intact over all portions of the tongue. There was diminution of sensation of pain, touch and temperature over the area supplied by the right auriculotemporal nerve. The patient was given an apple to eat. In about twenty seconds redness appeared in the right auricle and on the skin anterior to it; in about sixty seconds beads of perspiration were observed over approximately the same area. On another occasion the anterior two thirds of the right side of the tongue was swabbed with lemon juice. No reaction occurred. Then the posterior third of the tongue was swabbed. Sweating appeared promptly. By using Minor's method, photographs of the resultant condition were obtained (fig. 1). On another occasion the patient was given a hypodermic injection of pilocarpine hydrochloride; sweating appeared more profusely over the right auriculotemporal area than over the left, indicating hyperactivity of the parasympathetic nervous system. Furthermore, atropine, used therapeutically, produced a diminution in the sweating reaction, but the patient experienced discomfort from this form of therapy and refused it. It should be added that Stenson's duct was patent bilaterally. Especially after the injection of pilocarpine was the salivary secretion from the parotid gland visible.

COMMENT

The phenomenon of sweating over the cheek during eating has been recognized for many years. It was mentioned by Baillarger in 1847 but received the name auriculotemporal only as recently as 1923, when Lucie Frey so designated it. The

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From the Neurological Service of Dr. Israel Strauss, the Mount Sinai Hospital.

phenomenon occurs most commonly as a sequel to parotitis complicating an infectious disease, such as typhus or erysipelas. It occasionally occurs after traumatic lesions in the region of the parotid gland. In cases of infectious parotitis it makes its appearance about one month after the acute process has subsided. The syndrome consists of redness and sweating over the temporal and preauricular areas. The redness and sweating are not synchronous in appearance, the vasodilatation occurring first, nor are they conterminous in distribution; hence the probability is that the two reactions are not subserved by the same nerve fibers. In addition to the redness and sweating there may be pain in the same area during eating. Sensory disturbances, in the form of increased or diminished sensation in the distribution of the auriculotemporal nerve, are commonly present. Mere chewing will not evoke the reflex; mere tasting will. The relative efficacy of spicy foods

Fig. 1.—The dark area shows the result of the sweating test on the affected side.

in eliciting the response is most probably due to the fact, demonstrated by Pavlov, that spicy foods are particularly effective in stimulating the parotid gland. The fact that at least in one case after the entire tongue had been cocainized sweating could still be obtained has suggested that the afferent pathway must include the trigeminal as well as the glossopharyngeal nerve. Furthermore, it is of interest that in one instance the mere sight of a lemon being cut was sufficient to evoke the reflex.

Before proceeding to a consideration of the several theories postulated to explain the phenomenon, it will be helpful to review briefly the anatomic structures involved (fig. 2). The auriculotemporal nerve is a branch of the mandibular division of the trigeminal nerve. It passes through the parotid gland to reach the skin over the temporal region and over part of the auricle and the parotid gland. Before it reaches the parotid gland, it receives an anastomotic branch from the

otic ganglion. This branch comes from the glossopharyngeal nerve and proceeds as far as the parotid gland, supplying the gland with secretory impulses. The auriculotemporal nerve also receives a contingent from the cervical portion of the sympathetic trunk. Finally, the facial nerve supplies anastomotic branches to the auriculotemporal nerve. In addition to the glossopharyngeal innervation of the parotid gland, the auriculotemporal nerve therefore contains vegetative fibers from two sources: sympathetic and parasympathetic. I stress this point because it has been definitely established that sweating over the face depends on two different sets of fibers; after the cervical sympathetic ganglia have been extirpated pilocarpine is still effective in producing hidrosis.

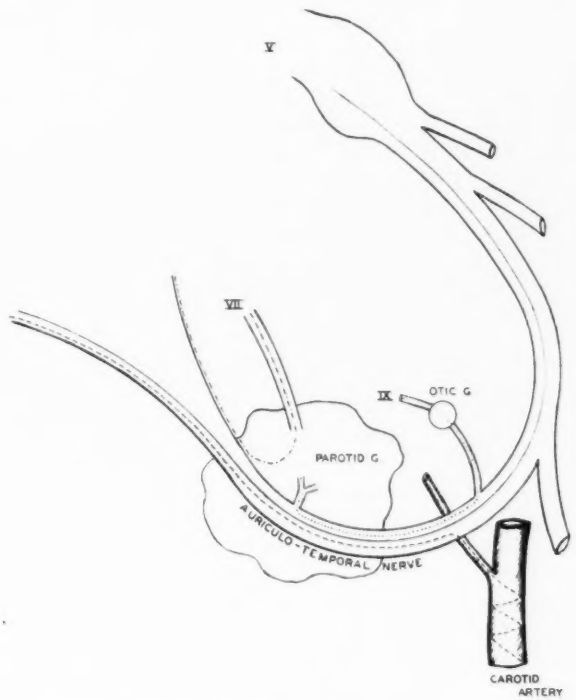

Fig. 2.—Diagram showing the formation of the auriculotemporal nerve (modified after Ford).

Like many other apparently simple phenomena encountered in neurologic practice, the auriculotemporal syndrome has given rise to diverse and complicated modes of explanation. There are essentially three theories proposed.

One view maintains that the phenomenon is merely an exaggeration of a reflex the pathway for which is physiologically always present. Reference is made to the occurrence of sweating over the face in otherwise normal persons while they are eating. The well known instance of Brown-Séquard, of the patient who perspired on both cheeks when he ate chocolate, is cited in this connection. Higier has attempted to describe the nerve pathways involved. The afferent arc is over the glossopharyngeal nerve and over the auriculotemporal nerve to a metameric autonomic center whence impulses proceed peripherally to the sweat

glands. Since in his case there was diminution in sensation over the distribution of the auriculotemporal nerve, he considered that the glossopharyngeal afferent arc assumed dominance of function and that therefore eating brought out the reflex. This explanation does not hold for cases in which sensation over the distribution of the auriculotemporal nerve is increased rather than diminished.

Another explanation is that after the auriculotemporal nerve has been injured during the parotitic process and while regeneration of nerve fibers is going on the glossopharyngeal fibers intended for the parotid gland are diverted from this pathway and instead either grow out into the sweat glands of the skin or anastomose with fibers intended to supply the sweat glands. Whether such a diversion of regenerating nerve fibers actually ever occurs has not, so far as I know, been demonstrated.

The third view appears to me most tenable, especially in the present case. This view maintains that as a result of the parotitic process scars form in the parotid gland about the nerve fibers. When the gland is stimulated into activity, it swells, and pressure is exerted on the vegetative fibers. This results in vasodilatation and sweating. It should again be pointed out that in the present case the phenomenon did not occur while the secretion of the parotid gland flowed freely through the fistula but did occur promptly after the fistulous opening had been sealed. A similar case in which closure of a fistula produced the syndrome is reported in the literature; likewise, a case of bilateral parotitis with a fistula on one side in which the syndrome did not appear on the side with the fistula but did appear on the side without one is recorded. These various facts lend support to the "parotid gland theory" of the auriculotemporal syndrome. It should be added that in the case reported here the sweating phenomenon appears to have been mediated, on the efferent side by the parasympathetic system, since the administration of pilocarpine produced greater sweating on the affected side whereas acetylsalicylic acid and heat yielded greater sweating on the unaffected side.

At the suggestion of Dr. Ira Cohen, radiotherapy was applied to the parotid gland. It was thought that if the third theory mentioned is correct, inhibiting the activity of the parotid gland should check the troublesome symptom. The therapy could not be continued beyond the third session, however, because of the patient's apprehensiveness. He feared that the dryness of the throat, which made its appearance after the third treatment, would become permanent if therapy were continued. As a matter of fact, the dryness disappeared shortly after the cessation of therapy, but the patient could not be prevailed on to submit to further treatment. Regarding the sweating phenomenon, the patient stated definitely that it became less marked after the third treatment and remained so for about two days. If his observation was correct, and it must be remembered that the statement was made by a man who was not overenthusiastic about the therapy employed, it would argue in favor of the theory proposed, namely, that the sweating is due to swelling of the parotid gland during its secretory activity with resultant pressure on the autonomic nerves. At any rate, this form of therapy deserves trial in future cases of the auriculotemporal syndrome.

NEW METHODS OF MEDICAL TREATMENT OF SCHIZOPHRENIA

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Ignorance of the pathobiologic processes underlying schizophrenia renders therapy of the etiologic factors as yet impossible. There are, however, accidentally discovered correlations which may form the starting point of therapeutic efforts, as in the case of dementia paralytica, in which remissions coinciding with accidental fevers led to the establishment of a biologic antagonism between the disease process and the febrile condition and thus to malarial therapy. Fever therapy has been applied also in cases of schizophrenia, but without results worth mentioning, owing, according to my conception, to the absence of a biologic antagonism between fevers and schizophrenia. My task was, then, to look for probable biologic antagonists of schizophrenia. Observations in this direction had been made before, without, however, being systematized and utilized for conclusions regarding therapy.

In 1929 Nyirö and Jablonszky published observations made on the epileptic patients of the Budapest-Lipótmezö Mental Hospital. They were struck by the fact that in cases of epilepsy combined with schizophrenia the epileptic convulsions ceased or became rare. In a case of epilepsy in which schizophrenia developed years after the onset, the epileptic convulsions became conspicuously rare and later ceased. The schizophrenic process itself subsided in this case in one year. Nyirö and Jablonszky reached the conclusion that the condition was cured in 1.05 per cent of cases of simple epilepsy, as compared with 16.05 per cent of cases of epilepsy complicated with schizophrenia—a statistical result proving the existence of a biologic antagonism between schizophrenia and epilepsy. This was recognized by these authors, who also made an attempt to utilize it therapeutically by treating epileptic patients with transfusions of blood from schizophrenic patients. The presence of this antagonism was stated later by G. Steiner and A. Strauss in a report on six thousand cases of schizophrenia. According to these authors, "typical epileptic convulsions are so rare in schizophrenia, if they occur at all, as to throw doubt upon the correctness of the psychiatric diagnosis." In 1930 G. Müller published reports of two cases of catatonia in which recovery ensued after spontaneous epileptic convulsions.

Summing up these observations, it is possible to establish the existence of a biologic antagonism between the schizophrenic and the epileptic process. I shall now report my experiments regarding the therapeutic applicability of this antagonism.

OBSERVATIONS

As already reported,[1] I endeavored to produce epileptiform convulsions in patients with schizophrenia and to observe the effect on the schizophrenic process. There are several ways of producing epileptiform convulsions. First, I used a 25 per cent oily solution of camphor in intramuscular injections, raising the dose from 8 to 30 cc. Later I used metrazol in a 10 per cent solution, injecting it intravenously in doses of from 3 to 6 or 7 cc. The difference between the ways in which these substances act is that after the administration of camphor the

1. de Meduna, L.: Ztschr. f. d. ges. Neurol. u. Psychiat. **152**:235, 1935.

epileptiform convulsions appear in from one to two hours, while after the administration of metrazol they appear immediately. Of the two, metrazol is the more suitable for the production of epileptic convulsions. The convulsions caused by the two substances are similar. As yet, however, I am in no position to judge whether the remissions caused by these substances are of equal value.

In my first publication I reported on twenty-six patients treated in this way. Of these twenty-six patients, ten were cured and three improved to the extent of being able to return to their normal surroundings; in thirteen the condition remained unaltered. The number of cases has since risen to forty-three, in which nineteen patients were cured, seven showed improvement and the condition of seventeen remained unchanged. In the wards for female patients (medical superintendent, Dr. A. Z. Ráth) similar experiments have been carried out with camphor only. Of thirty-one patients there, ten were cured, four showed improvement and the condition of seventeen remained unchanged. Summing up, Dr. Ráth and I have treated altogether seventy-four patients, of whom twenty-nine (39 per cent) were cured, 11 (15 per cent) showed improvement and the condition of thirty-four (46 per cent) remained unchanged.

COMMENT AND CONCLUSIONS

During the experiments I observed that the likelihood of cure is proportionate to the patient's liability to convulsions, as shown by the fact that the first ten male patients cured had, on an average, six convulsions after receiving altogether on an average 70 Gm. of pure camphor, while the first ten patients whose condition remained unchanged required a total dose of 129 Gm. on an average to produce an average of two convulsions. For a full valuation of the number of cures it is necessary to keep in mind that the material consisted mostly of patients with chronic conditions, chiefly of two to three years' duration. I am convinced that experiments carried out in a larger number of recent cases of conditions of not more than six months' duration would lead to considerably better results. These results do not affect the theory of the endogenous nature of schizophrenia. The number of cures—39 per cent—is rendered even more considerable when it is remembered that the condition in from 20 to 25 per cent of the seventy-four patients treated first, can be considered to be of hereditary nature, in which case the possibility of cure is out of the question.

The short duration of the experiments (one year) has prevented me from drawing far-reaching conclusions. Some of the cures may be due to an incidental spontaneous remission coinciding with the treatment. There are, however, two points to be emphasized:

1. The percentage of cures far exceeds the number of spontaneous remissions recorded in the literature.

2. There were relapses in which the prompt application of convulsive therapy led to a remission on the day following the convulsion.

The manner in which the convulsions may lead to therapeutic effects may be explained in different ways: First, I thought of the known detoxicating effect of camphor. This possibility, however, is excluded by the similar results achieved with the chemically entirely different metrazol. The shock effect of the convulsion may serve as an explanation. Easy as it may be to conceive that a catatonic stupor may be ended by a sudden shock, it is not plausible to imagine a cure of delusions and hallucinations in this way.

The only possibility left to explain the effect of the epileptic convulsions is that they change the chemical constitution of the organism in a way suitable for the cure of schizophrenia. Similar remissions of schizophrenia are not unknown, e. g., remissions preceding death, explainable by nothing but a preagonal change in the chemical milieu of the organism at the beginning of dissolution.

Far from considering the convulsion therapy as the only possible way of curing schizophrenia, I think that it is only a first step on the as yet unaccustomed biologic road of influencing the schizophrenic process. It is necessary to try other substances, as well as to search for the factor mobilized by the convulsion and causing its beneficial effect. In this way it will perhaps become possible to eliminate the convulsion and apply only its secondary effects.

News and Comment

NATIONAL COMMITTEE FOR MENTAL HYGIENE

The Division of Psychiatric Education of The National Committee for Mental Hygiene invites physicians engaged in the teaching of psychiatry to take part in a three day discussion of the content and methods of instruction. This conference will be held at the Henry Phipps Psychiatric Clinic, Baltimore, on April 8, 9 and 10, under the general direction of Dr. Adolf Meyer. Clinics and demonstrations will be a part of the program now being prepared by the division.

Abstracts from Current Literature

Anatomy and Embryology

A Study of the Occipital Region of the Chinese Brain. I. C. Wen, J. Comp. Neurol. **57**:477 (June) 1933.

The material consisted of seventy fetal brains of Chinese ranging from the fourteenth week to term. Fifty adult brains were used to check the findings in the fetal brains. Twelve specimens were used for histologic study, mainly to determine the distribution of the area striata. According to Cunningham, the precursors of the parieto-occipital and calcarine fissures are seen by the third month. In the present study, it was found that one or both may be obliterated during the fourth to the fifth month, depending on growth antagonisms. But by the seventh month of fetal life the typically shaped arrangement is permanently established. Histologically, the calcarine cortex has by this time fully attained all the characteristic features. The differentiation of the cortex appears first in the anterior calcarine sulcus and then proceeds in a caudal direction. On the lateral aspect of the occipital region, the lunate sulcus is first seen on the left hemisphere of a 28 week fetus. The variability in size, position and shape of the lunate sulcus is as evident in the brain in the fetal stages as in the adult brain. Seldom is the sulcal pattern symmetrical in both hemispheres. The lunate sulcus is recognized more often on the left than on the right side. Another observation concerns the shape of the calcarine fissure. It usually has a horizontal direction and may be gently curved, but in over half the brains studied it showed an angular bend, or hooklike form. This is confirmatory of van Bork-Feltkamp's description of the brains of Northern Chinese in which she found a calcarine fissure of this type in nearly half of the brains studied as compared with an occurrence of 18 per cent in the brains of the Dutch.

Addison, Philadelphia.

On the Size of Man's Brain as Indicated by Skull Capacity. Gerhardt von Bonin, J. Comp. Neurol. **59**:1 (Feb.) 1934.

Von Bonin has been able to remeasure several series of skulls, the study of which has been reported previously by different investigators. The skulls of males alone were considered. Von Bonin's results from direct determination of capacities were sufficiently different from the original results to cause him to state that direct determination of capacities should be used with caution. He believes that Hooke's formula is trustworthy. He concludes that while the human brain is larger than that of subhuman ancestors, no further increase has taken place since the time of Neanderthal man and that there is a definite indication of a decrease, at least in Europe, within the last ten thousand or twenty thousand years. The mean capacity of the skulls of Europeans is not higher than the interracial mean. Races of low civilization show very large as well as very small capacities. The intraracial distribution of cranial capacity is satisfactorily described by the normal law of errors. These facts suggest to him that size of the brain has become stable, and internal organization rather than bulk appears now to be of positive selective value.

Fraser, Philadelphia.

Some Observations on the Early Development of the Vestibular Nuclei in the White Rat. George A. Walker, J. Comp. Neurol. **59**:29 (Feb.) 1934.

The aim of this study is to supply a brief account of the developmental history of the vestibular nuclei of the white rat and to establish the sequence and the time

at which the more significant histologic and morphologic changes appear. A graded series of fetal and new-born rats was available after a variety of treatments. The first indications of the vestibular nucleus were seen in 15 day rat fetuses. Dendrites and Nissl substance were seen on the sixteenth day. Deiters' nucleus was seen to be derived from the lateral zone of the primitive cell mass by a process of proliferation and cellular differentiation. The superior vestibular nucleus developed from the superior and the lateral part of the primitive mass by a process of proliferation and migration of cells along the medial aspect of the restiform body, accompanied by cellular differentiation. These two nuclei were both distinguished by the sixteenth day of fetal life. The medial and descending nuclei developed simultaneously from the medial and the posterior region of the original mass of neuroblasts and could be distinguished from the fifteenth day. The central process from the cells in the vestibular ganglion reached the dorsolateral aspect of the medulla on the fifteenth day. Thus the anatomic relation of the vestibular ganglion and nuclei is established. On the seventeenth day bundles of fibers may be traced from Deiters' nucleus into the medial longitudinal fasciculus of the same and opposite sides, and from Deiters' nucleus into the restiform body. At this time a large bundle of fibers was seen extending from the superior vestibular nucleus superiorly and medially into the medial cerebellar nuclei. Thus the anatomic relations of the vestibular nuclei are established at least four days before vestibular function has been demonstrated.

FRASER, Philadelphia.

THE HYPOTHALAMUS OF NECTURUS. C. JUDSON HERRICK, J. Comp. Neurol. **59:** 375 (June) 1934.

This report is supplemental to a previous larger work on the forebrain of the Necturus in which the gross and internal structure has been described. The hypothalamus of the amphibian is separated by the sulcus hypothalamicus into dorsal and ventral parts. The dorsal part is the primordial mamillary body and is intimately connected with the ventral part of the thalamus and the cerebral peduncle. The ventral part as a whole is comparable with the mammalian tuber cinereum and infundibular region and is very intimately related with the preoptic nucleus which occupies the extensive area between the anterior commissure and the optic chiasm. The pars dorsalis hypothalami receives from in front fibers of the nervus terminalis and the larger part of the dorsal fascicles of the medial bundle of the forebrain. Thus it receives olfactory, visceral and gustatory excitations, all of which have been more or less influenced by other types of sensibility in the correlation centers before reaching the hypothalamus. This area is also connected by the tractus thalamomamillaris and the fibers of the postoptic commissure systems with the ventral hypothalamus and ventral thalamus. From these connections it is evident that the dorsal part of the hypothalamus is a premotor field into which fibers converge from a wide variety of correlation centers. The dominant sensory systems represented seem to be olfactory, gustatory and visceral. The chief outlet from the dorsal part of the hypothalamus is the mamillopeduncular tract. The ventral part of the hypothalamus has a massive afferent connection directly with the anterior olfactory nucleus. Efferent paths go rostrad into the medial bundle of the forebrain and caudad into the mamillary field and cerebral peduncles. The tractus hypophyseus is also described. This tract in Necturus is related with the gray substance of the preoptic nucleus and the ventral part of the hypothalamus and with the pars nervosa of the hypophysis. It is probably both afferent and efferent in function.

FRASER, Philadelphia.

THE DEVELOPMENT OF THE NUCLEI AND TRACTS RELATED TO THE ACOUSTIC NERVE IN THE PIG. RALPH F. SHANER, J. Comp. Neurol. **60:**5 (Aug.) 1934.

This is a study of the development of the nuclei and tracts of the acoustic nerve as seen in pig embryos impregnated by silver methods or stained by cytologic

methods. About 150 serially sectioned preparations were available. A general sensory nucleus for the fifth, seventh and eighth nerves is recognized in 16 mm. embryos. This breaks up into sensory nuclei for the spinal tracts of the vestibular and trigeminal nerves and for the tractus solitarius in a 24 mm. embryo. The cochlear nuclei are derived from the rhombic lip, which begins a striking proliferation in 16 mm. embryos and forms a thick stratum. The ventral cochlear nucleus is delimited in the 35 mm. embryo. The dorsal nucleus grows to a larger size and differentiates somewhat later than the ventral nucleus. The other nuclei of the acoustic system differentiate in situ out of the reticular substance. The superior olive appears when the embryo reaches the 24 mm. stage and the accessory olive, when it reaches the 100 mm. stage. At 150 mm. all the nuclei have adult cell characteristics. The primitive acoustic nerve is entirely vestibular and is seen in an 11 mm. embryo. Ipsilateral and contralateral secondary neurons of the vestibular nucleus are also seen. In 18 mm. embryos the semicircular canals differentiate. One full turn of the cochlea has grown in 30 mm. embryos. The cerebellum is growing rapidly. The ventral cochlear system of trapezoid fibers appears simultaneously with the cochlear nerve and nuclei. The acoustic striae appear later.

ADDISON, Philadelphia.

OBSERVATIONS ON THE PERIPHERAL COURSE OF THE SENSORY FIBERS IN THE FIRST FOUR CERVICAL NERVES OF THE CAT. J. C. HINSEY and K. B. CORBIN, J. Comp. Neurol. **60**:37 (Aug.) 1934.

Various dorsal root ganglia of the first four cervical nerves were removed aseptically in sixteen adult cats. After from ten to twenty-two days the peripheral and central nerve tissue from this area were treated by the Marchi technic. The spinal accessory nerve failed to show any evidence of degeneration after this treatment. No evidence of a contribution of sensory fibers from the dorsal root ganglia of the first four cervical nerves to the hypoglossal nerve was seen. The left nodose ganglion of the vagus nerve was removed in one cat. No degeneration was seen in either the intracranial or the muscular portion of the accessory nerve nor in the peripheral portion of the hypoglossal nerve. The authors conclude that the spinal accessory and the hypoglossal nerves in the cat do not contain proprioceptive fibers with cells of origin in the dorsal root ganglia of the first four cervical nerves.

ADDISON, Philadelphia.

ABERRANT PYRAMIDAL FASCICLES IN THE CAT. ROY L. SWANK, J. Comp. Neurol. **60**:355 (Dec.) 1934.

Variations in the course of the pyramidal tract are not as common in the cat as in man. In five cats studied in serial preparations stained by the Marchi method after the motor cortex had been excised, only one showed aberrant and recurrent pyramidal fascicles. The normal corticospinal tract could be followed as far as the lumbosacral region. Above the level of the inferior olive a lateral group of fibers was seen to shift laterad and to come to lie on the periphery of the olive. There it divided into two groups. One of these continued caudad into the spinal cord as far as the first lumbar segment. This is described as a homolateral descending ventrolateral tract. The other group encircled the olive and turned craniad to enter the restiform body, where its termination could not be followed because the cerebellum had been removed. This is a homolateral circumolivary recurrent fascicle. At the level of the hypoglossal nerve a small number of fibers cross to the opposite pyramid, where they become contralateral circumolivary recurrent fascicles and contralateral descending ventrolateral tracts. A homolateral ventral corticospinal tract was followed to the second thoracic segment of the cord. In the five cats a few fibers were recognized as a homolateral lateral tract. The only abnormality not found was a contralateral ventral corticospinal tract.

FRASER, Philadelphia.

THE EFFECTS OF ULTRACENTRIFUGING THE SPINAL GANGLION CELLS OF THE RAT, WITH SPECIAL REFERENCE TO NISSL BODIES. H. W. BEAMS and R. L. KING, J. Comp. Neurol. **61**:175 (Feb.) 1935.

This is one of a series of studies on the effect of ultracentrifugal force on various cell structures. Spinal ganglion cells of the albino rat were used for the experiments. They were put into Locke's solution and into the 1⅛ inch (2.86 cm.) all metal rotor which was driven by an air pressure of approximately 100 pounds (45.4 Kg.). A centrifugal force 400,000 times that of gravity was applied for ten, twenty, thirty or sixty minutes. The ganglia were then fixed and stained for microscopic study. The cells of the spinal ganglion often showed a marked displacement of many of their nuclear and cytoplasmic structures. The nuclei were stratified into three distinct layers from the centrifugal to the centripetal pole. The nucleus became slightly elongated and somewhat dumb-bell-shaped during the process of centrifugation. The Nissl bodies were readily moved to the centrifugal pole of the cell, which indicates that they have a greater specific gravity than any of the other cellular substances. Even after having moved through the cytoplasm, they often retained their discreteness. Not all cells react to the centrifugation in the same degree. The authors attribute this difference to variations in their physical consistency.

FRASER, Philadelphia.

Physiology and Biochemistry

BLOOD PRESSURE AND PULSE RATE: THEIR RELATIONSHIP TO CONVULSIONS AND THE FREQUENCY OF CONVULSIONS UNDER THE INFLUENCE OF OXYGEN INSUFFLATION IN CRYPTOGENIC CONVULSIVE STATES. J. NOTKIN, Am. J. Psychiat. **91**:147 (July) 1934.

The effect of the subcutaneous insufflation of oxygen was studied in thirteen persons with epilepsy. The oxygen was given into the thigh twice a week. The initial insufflation in each case was one of 500 cc., and subsequent ones of 1,000 cc. No definite correlation was noted between the occurrence of a seizure and the direction of change in the curve for the blood pressure between consecutive readings. For instance, seizures occurred as often when the blood pressure was stationary as when it was variable, and the change in the blood pressure was an increase as often as a decrease. The pulse rate varied, but the variations were in both directions. In only one of the thirteen cases was there a decrease in the number of convulsions during the experimental period, which in this case lasted for seven months. This patient had, on the average, five seizures a month before the insufflation, while during the experimental period, which lasted seven months, he had a total of only five seizures, or an average of less than one seizure a month. In two instances the frequency of the spells increased during the period in which insufflations were given—a fact which the author is inclined to ascribe to the withdrawal of the sedative or antispasmodic rather than to the effect of the oxygen. In the remaining five cases no significant change in the frequency of the convulsions was noted.

DAVIDSON, Newark, N. J.

CORRELATION BETWEEN THE DEVELOPMENT OF LOCAL REFLEXES AND REFLEX ARCS IN THE SPINAL CORD OF CAT EMBRYOS. WILLIAM F. WINDLE, J. Comp. Neurol. **59**:487 (June) 1934.

This report deals with a correlation between the development of reflexes and that of reflex arcs in 23 day old cat embryos. Spontaneous movement in 23 day embryos is rarely elicited by any means other than stimulation with the faradic current. The earliest spontaneous movements appear on the twenty-fourth day in an embryo from 14.5 to 15 mm. long. Two litters of known age, 23½ days, were available for the study of the reflexes. All of one litter gave definite reflexes of the foreleg, but none of the other litter did. All embryos were sectioned in a plane nearly transverse to the long axis of the cervical portion of the spinal cord. A

very close correlation was seen between the time of appearance of these local movements of the foreleg and the time of appearance of the sensory collaterals of the spinal cord. A series of unisegmental reflex arcs was demonstrated at the time reflexes appeared. The author emphasizes that the present observations have been confined mainly to the development of movements of the limb. He concludes that somatic movements appear in the forelimbs of cat embryos in the early part of the twenty-fourth day or at the end of the twenty-third day after insemination and that they appear to be true local reflexes. Correlated with the appearance of these movements, the first reflex arcs have been demonstrated in the spinal cord. They are homolateral, unisegmental, two and three neuron arcs found principally in the segments in the upper part of the brachial region.

ADDISON, Philadelphia.

THE RESULTS OF UNILATERAL PYRAMIDAL SECTION IN THE RAT. DONALD H. BARRON, J. Comp. Neurol. **60**:45 (Aug.) 1934.

To determine the function of the pyramidal tract in rats, this tract was sectioned in one of the pyramids in ten animals. The gait of the animals was observed from three to five hours after the operation until twenty days after, when the animals were killed. The brain and spinal cord were treated by Knower's modification of Cole's eosin and hematoxylin stain. The Marchi method is unsuitable for rat material because of the interrupted myelinization of the tract. The operation resulted in paresis of the flexor muscles of the limbs on the side opposite the lesion. This paresis disappeared in part during the first fourteen to twenty days. The residual deficiency was more easily observed in the forelimb than in the hindlimb. After complete section of the pyramid it has not been possible in any of the rats tested to elicit movements of either the ipsilateral or the contralateral limbs by bipolar faradization of the hemisphere on the side of the lesion. Movements were always to be elicited from the opposite hemisphere. The author concludes that the corticospinal fasciculus in the rat is concerned chiefly with the regulation of flexor movements of the forelimbs, especially of the digits and manus.

ADDISON, Philadelphia.

STUDIES ON CEREBELLAR FUNCTION IN THE TELEOST: I. REACTIONS RESULTING FROM CEREBELLAR ABLATION. II. IS THERE A CEREBELLO-TECTAL PATH? MARCHI METHOD. H. TUGE, J. Comp. Neurol. **60**:201 and 225 (Oct.) 1934.

This study consists of further observations on changes in dynamic or static positions after cerebellar extirpation. The efferent paths were determined by means of preparations stained by the Marchi method. Carassius auratus was used for the experiments. The operations consisted of total, unilateral or superficial removal of the corpus cerebelli. In control experiments both labyrinths and both optic lobes were removed. The stages of recovery after operation were classified as (1) traumatic period, characterized by irritation from operative traumatism; (2) deficiency period, which represents deficiency in behavior owing to the absence of the cerebellar function and to weakness, and (3) compensation period, in which the animal begins to recover by compensating for absence of the function. The fish that were subjected to operation exhibited altered behavior either at rest or in motion. Immediate reactions were those of rapid, spiral, up and down and disordered movements. While swimming, the body rolled from side to side. While resting, it either rolled or lay on its side on the bottom of the aquarium. Lowered tonus of the body musculature and rolling of the body persisted for over three weeks. Immediate reactions of unilateral decerebellate fish are like those of the total decerebellate fish, but the symptoms are less marked. The fish has a tendency to roll toward the operated side. The fish sometimes lay on its side with the body bent in a bow. After both total and unilateral decerebellation the movement of the eyes occurred much more frequently. Fish with superficially extirpated cerebellum showed functional deficiency, though to a lesser degree. Lesions made by cutting along the midline caused only slight reactions. In general, the more

extensive the lesion the more marked was the reaction, which suggests that there is no specialized functional center within the cerebellum. After extirpation of the labyrinth, the control fish was wholly unable to control its orientation. After both optic lobes were removed the fish swam normally without rolling movements.

On the basis of his experiments, Tuge concludes that the teleostean cerebellum has to do fundamentally with the postural mechanism of bodily muscle tonus as a whole. Specifically, the cerebellar deficiency causes the observed phenomena by interaction of the lowered muscle tonus and motor incoordination.

Thirteen of the animals used for the physiologic study of the cerebellum were kept alive for a sufficient time to allow the brains to be studied by the Marchi method. In addition to the crossed and uncrossed tractus cerebellotegmentalis, there were recognized cerebellifugal fibers to the tectum opticum, the tractus cerebellotectalis. This tract is first seen in close proximity to the tractus cerebellotegmentalis. Then it joins the lateral lemniscus on its own or the opposite side and proceeds rostrally with it. The exact locus of origin of these fibers was not established, but since the fibers are more abundant when only the more superficial layers of the cortex are destroyed the author believes they have their origin in a layer deeper than the Purkinje cells. The fibers end in the stratum medullare profundum of the tectum.

ADDISON, Philadelphia.

THE PATTERN OF CORTICAL INJURY IN THE RAT AND ITS RELATION TO MASS ACTION. NORMAN R. F. MAIER, J. Comp. Neurol. **60**:409 (Dec.) 1934.

The purpose of this investigation was to determine the deterioration in behavior resulting when various patterns of cortical tissue were destroyed. To test this deterioration, rats were tested for what Maier calls "reasoning ability" before and after operation. Animals that had been subjected to operation were divided into three groups: (1) those with one pair of trephine openings, (2) those with two pairs of openings and (3) those with three pairs of openings. Histologic examination revealed that the range and average amount of tissue destroyed were similar in the three groups and that the cortical areas covered were the same. Within each group there is a relationship between the amount of destruction and the postoperative score. The greater the destruction, the lower is the score. But the amount of destruction is not the only factor determining the degree of deterioration. Rats were grouped further into those without subcortical injuries and those with subcortical injuries. The latter group made poorer scores but as their cortical lesions were larger Maier concludes that differences in subcortical injuries between the groups were not responsible for the different results. The ratio of the length of the lesion to its width seems to be important, and deterioration seems to be the function of concentration of a lesion. Analysis revealed that when the amount of cortical destruction was constant there was a difference in deterioration in reasoning ability which was proportional to the ratio of the width of the lesion to its length. It is concluded that the pattern of a lesion as well as its mass is an important factor, determining the degree of resulting deterioration in behavior in the albino rat.

FRASER, Philadelphia.

THE BERGER RHYTHM: POTENTIAL CHANGES FROM THE OCCIPITAL LOBES IN MAN. E. D. ADRIAN and B. H. C. MATTHEWS, Brain **57**:355, 1934.

Hans Berger (1929-1933) first described a remarkable electric effect which can be detected in the human subject by means of electrodes applied to the head. This effect consists of a rhythmic oscillation of potential with a frequency of about ten a second, appearing when the subject lies quietly with eyes closed and disappearing if the attention is fully occupied. Berger concluded that the potential changes are produced by the cerebral cortex and that they represent a fundamental activity of the brain.

Adrian and Matthews state that in the consideration of the evidence as to the origin of the potential waves, the visual mechanism must be carefully considered. The Berger rhythm is obviously connected in some way with the visual mechanism,

and in all the conditions in which it appears, there is a lack of ocular fixation. If the Berger rhythm were due to clonus or tremor of the orbital musculature, active and passive movements of the eyeballs should give corresponding potential waves. This is not so. Also the potential gradients would be at a maximum in the neighborhood of the eye. However, the waves of the Berger rhythm are at a maximum over the occipital parts of the skull. The possibility of retinal potentials of some kind as the cause of the rhythm was eliminated when it was found that electrodes placed on the scalp could not even pick up the potential changes caused by illuminating the eye suddenly with bright light. The muscles of the scalp, face and neck as possible causes of the rhythm are eliminated. Berger rejected the possibilities of vascular pulsations, activity of the pilomotor muscles and tremors of the head as causes of the rhythm.

Positive evidence in favor of the cortex as the origin of the potential is derived from subjects with openings in the skull. Berger found that the potential waves are always larger when the electrodes are placed over the bony defect, wherever it may be. The observations of Adrian and Matthews in trephined patients lead to the same conclusion.

The magnitude of the potential waves show that they are most probably due to the simultaneous or nearly simultaneous activity of a fairly large region on the surface of the occipital lobe.

The failure of rhythm to occur during visual activity is due to the perception of pattern or to the attempt to perceive. It is not necessary that the eyes remain closed to produce the rhythm, for when the subject's eyes are open and the field of vision is uniform the rhythm makes its appearance, although it appears after a longer interval than if the eyes were closed. The rhythm can be abolished by a conscious effort to see even though the eyes are shut. This is also true when the eyes open in a uniform field. The perception of light does not affect the rhythm. Absolute uniformity of the visual field is not essential, and when the eyes are open the peripheral part of the field may contain some pattern. Sharp contrasts near the central part of the field abolish the rhythm. The rhythm is at its best immediately after the eyes have been opened and closed again, the most favorable conditions being those which exist immediately after pattern vision ceases. If the eyes are kept closed for a long time, the rhythm becomes intermittent. Examination of three blind patients failed to disclose any sign of the rhythm. This is to be expected in that pattern vision had ceased for these patients many years before.

As for nonvisual activity, a problem which claims the whole attention abolishes the rhythm, and so does an unexpected touch by the examiner. It must be kept in mind that a great deal can go on in the subject's mind without disturbing the rhythm, provided the content of thought is of a familiar nature and does not engage the entire attention of the subject.

The rhythm is explained as the result of a synchronous activity of a considerable number of neurons in the occipital lobe. This synchronous activity of a group of neurons can occur only when the conditions of excitation are uniform throughout the group. The abolition of the rhythm by pattern vision represents the change from synchronous to asynchronous action; this would follow from the disturbing effect of a mosaic of excitations in the optic tract.

The exact location of the beating area has not as yet been determined. All that can be said at present is that the information obtained from the study of the exposed brain does not conflict with the view that the rhythm arises in the occipital cortex.

The mechanism of the Berger rhythm as described by Adrian and Matthews is supported by their discovery that regular potential waves at frequencies other than ten a second can be induced by flicker. If a group of cells tends to beat spontaneously it should be possible to induce a beat at a higher rate by rhythmic stimulation. This is found to be so on exposing the eyes to a flickering field, and rhythms as high as twenty-five a second were obtained. When the frequency of the flicker is about ten a second it is extremely difficult to obtain a regular series of waves, there being periods of complete irregularity. With careful adjustment it may be possible to make the normal rhythm and the flicker coincide. Once

the frequency of ten a second is established for the flicker, the normal rhythm is able to resist disturbing influences which would upset the spontaneous beat. The greater stability of the response to the flicker is natural enough: With a flicker a coordinated beat is imposed on the area by the rhythmic excitation, whereas with the spontaneous waves there is nothing but their own interaction to synchronize the different neurons.

Adrian and Matthews' view on the rhythm differs from that of Berger, who believed that the rhythm represents the normal activity of the entire cortex. Their evidence points to the occipital lobes as the seat of the rhythm.

SALL, Philadelphia.

THE FLEXION REFLEX AND ITS CLINICAL SIGNIFICANCE. J. ROTHFELD, Rev. d'oto-neuro-opht. **12**:648 (Nov.) 1934.

The flexion reflex (called "reflex of the leg on the leg" by Balduzzi) is described as a flexion of the thigh in the coxofemoral articulation and a flexion of the leg in the articulation of the knee, with dorsal flexion of the foot and toes, produced by performing on the other extremity a passive flexion of the coxofemoral articulation with the leg in extension or flexed on the thigh. Rothfeld has observed this phenomenon in fifty-four cases, including thirty-seven cases of tumor of the brain, one case each of cerebellar abscess, hydrocephalus and serous meningitis and fourteen cases of vascular lesions in the brain. The reflex occurs on the same side as the lesion of the brain or on the opposite side and has been observed on both sides in a case of abscess of both hemispheres. In cases of a lesion of the frontal lobe located in front of the knee of the corpus callosum the reflex occurs on the side opposite the lesion; in cases of a lesion situated behind the knee of the corpus callosum and of those which end in hemiplegia the reflex is on the same side as the lesion; in cases of cerebellar tumors it is found on the same side as the lesion. It has not been observed in cases of lesions limited to the temporal lobe and not encroaching on the motor pathways.

The localizing value of this sign is limited. The presence of the flexion reflex, may, however, facilitate the differential diagnosis of lesions of the anterior part of the frontal lobes, and when the evidence points to a lesion of a given side of the brain it can determine whether the lesion is in front of or behind the knee of the corpus callosum. In cases of cerebral diseases accompanied by hemiplegia the occurrence of the reflex on the side of the lesion is the rule. In cases of recent hemiplegia following cerebral hemorrhage the reflex occurs on the sound side in the beginning and disappears as the condition improves. This same retrogression is observed also in cases of gumma of the brain, encephalitis and tumors accompanied by edema.

A hypothetic explanation of the mechanism of the retrofrontal flexion reflex is that the two cerebral hemispheres possess an excitatory influence for the reflex and that each hemisphere has normally an inhibiting influence on its fellow; if the function of one of the motor spheres is abolished, its inhibiting influence is suppressed and the opposite hemisphere is free to exercise its excitatory action, and thus the reflex appears in the opposite leg—on the side of the lesion. It is not known why the reflex is not observed in cases of lesions of the pyramidal bundles. The inhibiting influence of one hemisphere on the other is transmitted through the corpus callosum; consequently an isolated lesion of this body should result in the occurrence of the reflex on both sides, but no case of this kind has been observed. The propagation of the inhibitory action is along the frontopontocerebellar tract, cerebellum, nucleus ruber and the rubrospinal tract. Therefore, when this pathway is obstructed stimulations that arise in a frontal lobe pass to the leg of the opposite side. Rothfeld's conception warrants the presumption of the production of the flexion reflex on the same side as the lesion in cases of cerebellar lesions, and four observations support this view. Certain experimental researches appear to support this theory as do also the experiments of Hoff. In doubtful cases the presence of the reflex may be a valuable aid in guiding the diagnosis to a cerebral lesion.

DENNIS, San Diego, Calif.

VAGAL EFFECTS TRANSMITTED THROUGH THE BLOOD STREAM IN THE WHOLE ANIMAL. E. V. SAALFELD, Arch. f. d. ges. Physiol. **235**:22, 1934.

In dogs the vagus fibers to various organs (heart, lung and intestines) were stimulated, and the effect on other organs (salivary glands, bronchi and vessels) was studied. In order to preserve the acetylcholine-like substance produced by stimulation of the vagus fibers physostigmine was injected, and on stimulation of the abdominal vagus fibers the portal vein was directly connected with the vena cava (because the liver removes acetylcholine from the blood). It was found that stimulation of the cardiac vagus fibers induces increased salivation and contraction of the bronchi. On stimulation of the pulmonar vagus fibers increased salivation was also observed. The same effect and also a fall in the blood pressure and contraction of the bronchi were noted on stimulation of the abdominal vagus fibers.

SPIEGEL, Philadelphia.

ANALYSIS OF FATIGUE IN THE NERVE-MUSCLE PREPARATION. J. WALIDOW, Arch. f. d. ges. Physiol. **235**:147, 1934.

Nerve muscle preparations of frogs (Rana esculenta) were fatigued by indirect stimulation and were then perfused with Ringer's solution with a calcium content higher than normal. On continued stimulation an increase of the muscular contractions was observed, while the contractions diminished when the preparations were perfused in simple Ringer's solution. This effect of calcium is due to an action on the nerve endings. Calcium had no effect on the muscle itself or it had an inhibitory effect in higher concentration. It is concluded that the nerve-end apparatus in a fatigued nerve muscle preparation is in a state of "local fatigue" which is abolished by calcium.

SPIEGEL, Philadelphia.

THE EFFECT OF HUMAN BLOOD SERUM ON BRAIN TISSUE. J. VON BALO, Arch. f. Psychiat. **102**:202 (June) 1934.

Von Balo has undertaken a series of experiments to investigate the effects of different types of human blood serum on brain tissue. A 10 per cent emulsion of rats' brains was prepared in physiologic solution of sodium chloride and to 1 cc. of this emulsion 2 cc. of blood serum was added. This was placed in a thermostat set at 37 C. for twenty-four hours. The preparations were made under aseptic conditions. As controls, two preparations were used: brain emulsions without serum and brain emulsions to which normal blood serum was added. At the end of the twenty-four hours the amount of free inorganic phosphoric acid was determined and compared with the amount of this substance present before the serum was added. The serums of twenty-one normal persons and of a group of patients with different types of disease were used. The latter group consisted of eighteen persons with dementia paralytica and tabes, seventeen with syphilis, twenty-three with multiple sclerosis, fifteen with diseases of the central nervous system other than syphilis and twenty-eight with diseases in which the central nervous system was not affected. The results were: The serum of healthy persons does not materially affect the brain emulsion. The blood serum of patients suffering from multiple sclerosis, dementia paralytica and tabes gives rise to the appearance of considerable amounts of inorganic phosphoric acid; this, however, is not true in all cases; the effect of the serum from patients suffering from syphilis without involvement of the nervous system is similar to that from patients in whom the nervous system is affected. The serum of patients suffering from diseases not involving the nervous system shows an appreciable effect only in rare cases, such as those of purulent conditions and of diabetes. The effect of the serum of patients with dementia paralytica on the brain emulsion can be influenced favorably by treatment with malaria.

MALAMUD, Iowa City.

THE NEURON THEORY OF THE PERITERMINAL NETWORK OF BOEKE. S. RAMÓN Y CAJAL, Arch. f. Psychiat. **102**:322 (July) 1934.

Cajal discusses the publications of Boeke and some of his followers on the periterminal network which exists between the nerve-ends and the muscle tissue. He considers primarily the question whether this network is part of the nerves or some independent structure. A number of interesting experiments made by the author and by other workers are discussed, and Cajal concludes that although one cannot deny the objective reality of the network, doubt exists as to its belonging to the neurofibrils. From a physiologic point of view he considers this fact only as of secondary importance, because whatever structure this network may represent it certainly must function as an intermediary agent between the nerve and the responding muscle.

MALAMUD, Iowa City.

THE ELECTRO-ENCEPHALOGRAM IN HUMAN BEINGS (NINTH CONTRIBUTION). H. BERGER, Arch. f. Psychiat. **102**:538 (Nov.) 1934.

Berger discusses his technic in obtaining electro-encephalographic records, stressing particularly the facts that he uses silver needle electrodes which are well insulated to within a short distance of the point and that they are inserted under the scalp and periosteum in direct contact with the skull. One electrode is placed on the occiput and one on the forehead. In this way Berger obtains two types of waves, the large, slow "alpha" waves, and the shorter but faster "beta" waves. He considers the alpha waves as expressions of the function of the cortex, whereas the beta waves he regards as probably representative of the vegetative functions of the whole brain. The results discussed in this paper were obtained (1) in a normal person who was subjected to a decrease of oxygen concentration in the blood and (2) in an epileptic person in whom convulsions were produced by hyperventilation. In the experiment on the normal person it was observed that just about at the time the person lost consciousness and while the unconsciousness lasted there was a pronounced change in the alpha waves in that they became much larger and tended to occur in groups 0.44 second apart. This the author considers as an expression of the removal of inhibition of the spontaneous cortical activities by the lower centers such as the thalamus. A similar record was obtained in the experiment on the epileptic patient in the initial state of unconsciousness, induced by the slow deep respirations. After this phase and with the occurrence of the tonic phase of the convulsion, there was disappearance of the alpha waves. Berger discusses a theory considering the physiology and possibly the pathology of the epileptic convulsions in relation to the changes in the electro-encephalogram.

MALAMUD, Iowa City.

Psychiatry and Psychopathology

CONCERNING THE CAUSE OF DEATH IN CERTAIN PSYCHOSES. GERSON DAVIDSON, Am. J. Psychiat. **91**:41 (July) 1934.

Twenty-two patients who entered the hospital in a state of acute psychomotor excitement and who died shortly after admission were studied in order to determine the cause of death. No obvious physical illness was found in any case. Four of the patients suffered from the manic type and eighteen from the catatonic type of psychosis; all were females. They declined rapidly, the temperature fluctuating, the blood pressure falling, the leukocyte count rising and the weight diminishing. The faces were flushed; the heart beat was vigorous, but the volume of the pulse was small. At autopsy, congestion was found in almost every organ, especially in the brain, spleen, liver, meninges and kidneys. The intracranial fluid was increased. The small blood vessels generally showed degeneration and tortuosity.

Davidson concludes that capillary dystonia was present and that the patients suffered from a "capillary toxicosis." He is inclined to think that the toxic agency is similar to the "H" (histamine-like) substance of Lewis. As a result of this

condition the lumens of the visceral capillaries are widened, the widening producing congestion and handicapping regeneration, and eventually causing cardiovascular collapse. Hypothalamic dysfunction may account for the trophic changes and fluctuations in temperature.

HENRY A. DAVIDSON, Newark, N. J.

MARIAHUANA INTOXICATION. WALTER BROMBERG, Am. J. Psychiat. **91**:303 (Sept.) 1934.

The flowering tops of the hemp plant contain an active principle, cannabis, which is being used increasingly under the names "mariahuana," "hasheesh," "loco weed," etc., as an acute intoxicant. Its use was introduced into the United States at the seaport cities and along the Mexican border, and in the last few years it has become a problem in large cities of the country. Because of its tendency to release aggressive and sexual drives, the drug may have certain antisocial effects. In itself, however, mariahuana does not produce any actual ethical or moral degeneration, and the statistics on the association of addiction to cannabis with crime rest largely on the fact that inadequate and criminal persons are more likely to become chronic users of the drug, as well as on the fact that the preparation may release previously existing antisocial trends. Bromberg does not consider the drug genuinely habit forming, as no symptoms develop when it is abandoned and no real tolerance is developed.

When inhaled, mariahuana produces a brief period of anxiety, soon displaced by euphoria. In the latter stage talking becomes rapid, laughter is uncontrollable and the limbs appear to be light. Time is slow in passing, while activities appear to speed up tremendously. Visual hallucinations may developed. After an hour or two the subject falls into a dreamless sleep from which he awakens without physiologic after-effect.

Ten cases of mariahuana intoxication are reported. In two there were psychoneurotic reactions to periods of intoxication; in five there were toxic psychoses, and three were ordinary cases of acute intoxication. The intoxication appears to bring the individual personality forces and conflicts to the surface. The change in sense of time is a universal and characteristic feature of the intoxication. Time is normally measured subjectively by the regular recurrence of events, so that the increased speed of associations and perceptions and the large number of experiences which crowd on the observer give the impression of a stretching-out of time.

DAVIDSON, Newark, N. J.

THE ANAMNESIS OF THE TOXIC GOITER PATIENT. AGNES CONRAD, Am. J. Psychiat. **91**:521 (Nov.) 1934.

In taking the history of a patient with toxic goiter the interviewer generally notes that when an emotionally painful point is touched on, the coherence of the narrative is broken, confusion occurs and blushing, weeping, blotching or venous pulsations become apparent. Just as the surgeon maps out a tender abdominal area by careful palpation, so the psychiatrist can delimit an emotional pathologic process by noting the responses of the patient during the interview.

In a series of 200 cases of toxic goiter reviewed by Conrad, all but 8 indicated the existence of a distressing emotional episode immediately prior to the onset of the toxic symptoms. Further investigation of this event suggested that it represented a precipitation of an emotional situation to which the patient had become specifically sensitized in earlier life. In reviewing a series of cases by this technic, Conrad finds that the patient's relationship to his or her mother played a disproportionately large rôle in these earlier, sensitizing experiences; usually this took the form of identification with the mother, fear of losing her or dread of being unable to equal her. Conrad suggests that the emotional disturbance may upset both the endocrine and the vegetative balance. Psychotherapy should supplement, but must not replace, medical and surgical treatment in these cases.

DAVIDSON, Newark, N. J.

THE BRIGGS LAW OF MASSACHUSETTS. WINFRED OVERHOLSER, Am. J. Psychiat. **91**:585 (Nov.) 1934.

In Massachusetts the Briggs Law provides for the impartial examination of defendants repeatedly indicted for felonies by two psychiatrists assigned by the state department. The psychiatric report is not admissible in evidence, but it is forwarded to the court and attorneys, and the psychiatrists are available for oral testimony in court by the usual process. The right of either party to present his own psychiatric experts is preserved but rarely exercised. Between 1921 and 1933 3,610 defendants were examined; 61 were classified as insane, and 336, as mentally defective; 83 per cent of the subjects examined were classified as not abnormal. Nearly all of the subjects classified as insane were committed to hospitals for patients with mental disease, but only 11 per cent of those classified by the psychiatrists as mentally defective were committed to custodial institutions for the feebleminded; most of the others were sent to the conventional penal institutions. Overholser lists the merits of the Briggs Law as follows: (1) provision to the defendant of competent, impartial, psychiatric examination, (2) provision to the court and counsel of competent information, (3) avoidance of the disgraceful battle of experts, (4) saving of money for the state by avoiding futile trials of incompetent defendants, (5) discovery of many abnormal persons whose condition would otherwise have escaped diagnosis, (6) aid in educating the bar and the public and (7) protection of the rights of the mentally incompetent.

DAVIDSON, Newark, N. J.

SUICIDES AND HOMICIDES IN THEIR RELATION TO WEATHER CHANGE. C. A. MILLS, Am. J. Psychiat. **91**:669 (Nov.) 1934.

During periods of falling barometric pressure all persons are afflicted with a sense of futility, an attitude of pessimism and an inability to reach the usual level of efficiency. During periods of rising pressure, one tends to feel buoyed up and alert. During the periods of falling pressure one is irritable, quarrelsome and depressed, and during such periods suicide is most frequent, and the irritable, violent adult is impelled to impulsive assault and even murder. Mills presents maps to show the distribution of homicide and suicide rates and points out the similarity between these distributions and the distribution of movements of areas of low pressure throughout the country. The highest homicide rate in the country is recorded at Kansas City and at Memphis, where southern heat and northern storms meet and where tornadoes are most prone to occur. Along the southeast coast, where the weather is more stable than in the South generally, the rates for suicide and homicide for both the white and the Negro population are lower than in the rest of the South.

A day by day record of suicides and barometric pressure readings in a central area of the country was kept by Mills for five years. Analysis shows that there is a distinct relationship between falling barometric pressure and rising incidence of suicide. Repeatedly, sudden peaks in curves for suicides were found to coincide sharply with low pressure crises. The author thinks that weather is a factor of major importance in the problems associated with mental instability and that of all the weather factors, falling barometric pressure is most disturbing. Instability and violent change in the pressure rates have been more significant than a consistently low pressure.

DAVIDSON, Newark, N. J.

SOCIAL CONSCIOUSNESS. H. L. LATHAM, J. Abnorm. & Social Psychol. **29**:287 (Oct.-Dec.) 1934.

There are many theories concerning the forces which formulate the human psychic life. Among these are: (1) the assumption that the experiences and wishes of ancestors rule the psychic life, (2) Freud's view of the psyche as the arena of different impulses which, corresponding to various aspects of the sex impulse, combat each other, (3) the reflex theory of Bechterew which looks on the most

complicated behavior as simply the automatic unfolding of definite reflex mechanisms which are directed and fixed through habit and (4) Watson's behaviorism, which claims that man's acts and attitudes are a pure product of his environment. One thing is certain: that man belongs to the race of living creatures which cannot exist without close association with their fellows. He lacks the equipment to maintain himself alone in life. It has been observed that the character and actions of the single man are determined by the experience of society in which he grows. Though this seems to approach closely the views embraced by Watson, there is another factor of significance, namely, that similar experiences and impressions are assimilated variously by different persons. Man not only reacts, he takes a position. It is not the real environment that is reacting on the man but the environment as subjectively comprehended. Primarily through revolt or dependence and through defiance or surrender man develops his characteristic behavior—his character. This urge to adjust oneself to the definite conditions of the environment is represented in the social consciousness of every man. Human society sets three tasks before every person: occupation, social relations and love. These embrace the entire human life with all its aspirations and cares. Every human woe springs from the difficulties which arise from the attempt to perform these tasks. The possibility of success does not lie in the abilities of the person, nor in his intelligence; men with preeminent qualities fail; others with limited qualities succeed relatively. Subjectively, social consciousness expresses itself in the conviction as to social unity. This has an objective component—the degree to which the individual man shows himself a good comrade in life. But this objective compliance does not necessarily presume a real or subjective feeling for mankind, for a man may suddenly break down when he apparently was adapted to life. The cause of this may possibly be found in the fact that such a person had never really been socially conscious. Every one must sooner or later prove how far his social consciousness is developed.

WISE, Howard, R. I.

THE MENTAL HEALTH OF THE WORKERS. REX HERSEY, Ment. Hyg. **18**:462 (July) 1934.

From a psychologic study of twenty-nine American and fifty-two German workers, Hersey concludes that these laborers are happy only 55 per cent of the time. He also finds that a happy worker produces 2 per cent more than the general norm, while an unhappy worker produces 7 per cent less. Among the factors which have sufficient emotional tone seriously to influence the happiness and unhappiness of the workers, the author lists industrial factors (nature of work and supervision), physical condition of the worker, social factors (relations with friends and family, recreational influences, etc.), political problems, financial problems and weather. The relative influence of these factors varied in the two groups. In Germany, politics influenced 10 per cent of the workers' mental states, whereas its influence was only on 0.1 per cent in the United States. Strictly financial problems were likewise more influential in Germany, contributing to 10 per cent of the emotional state of the German, and to only 2 per cent of the American, worker. The industrial and physical factors, on the other hand, were more significant in the United States, while the social-domestic factor was slightly more influential in Germany. The weather's influence is evaluated at 7 and 8 per cent in the United States and Germany, respectively. Among the specifically important factors contributing to a worker's happiness were feeling that he was making progress toward a goal and knowing that his work was worth while and was recognized as such.

DAVIDSON, Newark, N. J.

STUDIES IN CATATONIA. CARNEY LANDIS and T. W. FORBES, Psychiatric Quart. **8**:745 (Oct.) 1934.

Patients suffering from catatonic dementia praecox are less likely to assume fetal postures during sleep than are normal subjects, while during the waking state

such patients are more likely to present a fetal position. This suggests that any theory which conceives of sleep as a regressive state and of catatonia as the expression of a wish to return to a more primitive status must be revised, for in catatonic subjects, in whom this should be most marked, the regression seems to be limited to the waking state. The authors are inclined to believe that these fetal postures are merely efforts to maintain bodily warmth.

Flexibilitas cerea disappears during sleep. From this two possible theories may be developed: (1) that the waxy posture is a more or less voluntarily controlled phenomenon based on some individual psychic idiosyncrasy, or (2) that the posture may be the result of a functional disturbance of the brain stem, which is neutralized or eliminated by some process of sleep—possibly by the activation of the sleep centers. In either event, neither toxemia nor any other protracted systemic condition can be reasonably blamed for the waxy flexibility.

The paper is a summary of six studies in catatonia previously reported.

DAVIDSON, Newark, N. J.

THE PREVALENCE OF MENTAL DISEASES AMONG THE URBAN AND RURAL POPULATIONS OF NEW YORK STATE. BENJAMIN MALZBERG, Psychiatric Quart. **9**:55 (Jan.) 1935.

Defining an urban community as one with a population in excess of 2,500, Malzberg finds that there is a lower incidence of mental disease among the rural than among the urban population. In cities the rate tends to vary more or less directly with the size of the community. To some extent this may be due to the fact that certain types of patients with mental disease are more readily cared for in country homes than in metropolitan apartments; but this consideration is inadequate to explain the full measure of the difference. The proportion of cases by which the urban rate exceeded the rural rate may be expressed as the number of urban patients to ten rural subjects. For the different psychoses this ratio is as follows: senile dementia, 20 (i. e., there are proportionately twice as many cases of senile dementia in urban as in rural areas); manic-depressive psychosis, 13; dementia praecox, 18; alcoholism, 20, and dementia paralytica, 26. When the statistics are examined separately for each sex, little difference in these proportions is noted, except for alcoholism, in which among females there were 31 cases in the urban population to 10 cases in rural districts. DAVIDSON, Newark, N. J.

WHAT HAPPENS TO MENTAL PATIENTS AFTER DISCHARGE FROM THE HOSPITAL? RAYMOND G. FULLER, Psychiatric Quart. **9**:95 (Jan.) 1935.

An analysis of a follow-up study of persons admitted to the New York State hospitals, made fifteen years after their first admission, shows that 42 per cent of patients had been discharged, 41 per cent had died in the hospital and 17 per cent were still in the institution. Fuller sought to determine the careers of the patients who had been discharged. The study shows that in ten years 21 of each 100 patients discharged had been returned to a hospital for patients with mental disease and 55 were living in the community. The remaining 24 were dead. Thus the proportion of discharged patients living in the community ten years later was 55 per cent; among males the incidence was only 51 per cent, and among females it was 57 per cent. The difference is due in part to the age factor. While more female patients remained out in the community, a higher percentage of the males was definitely successful. Thus, of the patients who had resided continuously outside the institution during the period of the survey, 72 per cent of the males and only 65 per cent of the females adjusted themselves with apparently real success. At the end of ten years, 20 per cent of the manic-depressive patients and 43 per cent of the schizophrenic patients were back in a hospital.

DAVIDSON, Newark, N. J.

THE PSYCHOANALYTIC THEORY OF PLAY. ROBERT WALDEN, Psychoanalyt. Quart. **2**:208, 1933.

In many cases a child's play seems to be an elaboration of material recently experienced, but these experiences are altered in the play in accordance with the pleasure principle in such a way that they repeat some pleasurable experience, indicating the fulfilment of some wish. Often, such wishes are a desire to be big and strong, as when a little girl plays she is a mother. Another type of wish-fulfilling play is seen when a child shortly after the birth of a sibling plays he is a baby. At times, however, the play is a repetition of a painful experience, such as a visit to the dentist or the physician. Bühler explained these different reactions by classifying play into two types: (1) functional pleasure play and (2) gratification pleasure play. Play of the first type is undertaken for the pure pleasure of action regardless of the kind of action or the success of the activity; play which repeats an unpleasant experience is placed in this classification. Play of the second type is undertaken for the gratification arising from the success of the activity. Bühler's theory of functional pleasure play does not explain the specific content of the play. A child who wishes this type of play may as well play at any activity, whereas visits to the dentist are frequently reenacted in the play.

The specificity of play seems to arise in this way: the child, frightened and hurt by the dentist for whom he has had to be the passive sufferer, has received a severe psychic trauma. The repetition compulsion of his id impulses forces him to repeat the experience, and his ego tries to reenact the situation as if he were the actor, thereby enabling himself to master and assimilate the unpleasant experience. In the play the child is no longer the frightened spectator, but rather the cause of the fear, and he behaves like the patient with war neurosis whose dreams are an attempt to assimilate and master the mass of excitation to which he has been suddenly exposed. For this reason children frequently play out experiences which are unpleasant and traumatic. Children are able to do this because they merge reality with fantasy at an early age. This is of practical importance, for if a teacher or parent knows that a child has been subjected to an unpleasant, traumatic experience, he may induce the child to repeat the experience in his play, making himself the active person. Children in their play may also enact rôles which are not permitted to them by parents or by the super-ego. Children's play differs from adult fantasies in that in the latter the fantasy is not about a real object, while in the former it is.

Walden summarizes the psychoanalytic theory of play as follows: It expresses the instinct of mastery; it has the purpose of a wish fulfilment; it has the aim of assimilating overpowering experiences by repetition compulsion and through the change from passivity to activity; it often allows a leave of absence from reality to the super-ego; it expresses fantasies about real objects.

PEARSON, Philadelphia.

FROM THE ANALYSIS OF A BED WETTER. ANNY ANGEL, Psychoanalyt. Quart. **4**: 120, 1935.

Angel reports the analysis of a girl, aged 12 years, who suffered from enuresis following a severe and mysterious illness characterized by violent spells of vomiting and possible fever when she was about 3 years of age. Before this she had been trained successfully in cleanliness. Analysis revealed that at that time, when her parents were away, she was staying with her grandmother and had made the acquaintance of a gipsy. The gipsy had lured her to his room and requested her to turn somersaults to expose herself. She had interpreted his laughter as ridicule of her genitals. He had then shown her his penis and had induced her to play with it and had played with her genitals. As a result of this she acquired the idea that her genitals were so defective that she could not control her urine, so that it must always flow out against her will. Urinary organs and genitalia were

closely associated in her mind. She believed she urinated from the vagina and that a man had two penises, a flaccid one for urination and an erect one for intercourse. Following analysis of this incident the enuresis ceased and remained absent for three years. The patient's mother interrupted treatment at this point, being satisfied with the clinical improvement, so that analysis was not completed.

PEARSON, Philadelphia.

INTROJECTION AND PROJECTION, GUILT AND RAGE. KARIN STEPHEN, Brit. J. M. Psychol. **14**:316, 1934.

This article is a formulation of the psychoanalytic point of view of symptoms due to errors of perception of reality in relation to the inner and the outer worlds of the subject. It is based on clinical material gathered from the study of infants and neurotic and psychotic patients. Confusion, it is stated, is the portion of infancy, and is overcome only by understanding gained through experience. Before distinction of the self and nonself has clearly developed, division is made into controllable and uncontrollable; good internal happenings and obedient external ones belong to the self; bad, unmanageable ones are regarded as an outside enemy, with fear and aggression. Originally nonsexual, this fear and aggression become secondarily libidinized. In persons with paranoia this childhood belief is conscious; in certain neurotic persons it is held in the unconscious through regression or fixation.

ALLEN, Philadelphia.

ANXIETY: ITS NATURE AND TREATMENT. HENRY HARRIS, J. Ment. Sc. **80**:482 (July); 705 (Oct.) 1934.

Harris attempts to coordinate the views of Pavlov and Freud in reference to major psychologic problems and especially to the problems of anxiety. He postulates four fundamental moods, which may be considered in terms of conflict: anxiety as unsolved conflict, elation as solved conflict, depression as unsolvable conflict and apathy as the most complete solution of conflict. Conflict may be considered in terms of the tensions interacting to cause conflict and the balance between such tensions. In anxiety the tension is a rising one; in elation it is a falling one; in apathy it is low and is continuously being discharged; in depression it is extremely and continuously high and amounts to a sort of spasm. These tensions may reasonably and profitably, if somewhat speculatively, be considered to be metabolic, visceral and autonomic tensions. Harris' plan of treatment is as follows: The problem at hand must be resolved clearly into terms of anxiety. This may be simple or may require the full resources of the psychoanalytic technic. An adequate outlet, not conflicting with social demands, must be found for the energy that is bound up in the anxiety, that is to say, for the segment of instinctive activity under greatest tension. Failing this, the subject must be "cotton-woolled" and spared anxiety-producing and tension-increasing stimuli. As a last resource, "supportive" treatment must be used to combat anxiety which is completely unavoidable. This, by diminishing the total energy of the patient, relieves the painful hypertension of individual segments or viscera.

KASANIN, Howard, R. I.

PSYCHOLOGICAL EFFECTS OF BODILY ILLNESS IN CHILDREN. DAVID FORSYTH, Lancet **2**:15 (July 7) 1934.

It is to be expected that children, in keeping with their sensitiveness to early experiences, will be affected psychologically by bodily illness, but it remains to be generally recognized that the dispositions of sick and invalid children may be subtly and profoundly changed in ways which may persist after convalescence and even throughout life. Since these changes are usually gradual and a child is hardly able to speak of them, they are not often understood and are rarely attributed

to their real origin. As it is beyond children to formulate scientific explanations of diseases, they fantasy their own interpretations. Very young children believe that illness, with its pain and discomfort, is caused by some living being gnawing and biting inside themselves. Later, and for many years, they regard illness as a punishment, which they suppose can supervene only in connection with some wrong they have done, and the feelings of guilt come into close relation with the notions about illness.

A frequent effect of a temporarily incapacitating illness is the retardation of the child's mental development and even the production in a younger child of an obvious regression of character and intelligence. This is accounted for in part by the restricted activities, as well as by the many things which, in Forsyth's opinion, seem to stimulate regression to the infantile stage. The child refamiliarizes itself with life in the crib; helplessness is increased and food is usually restricted to milk.

Fear not infrequently originates in illnesses associated with feelings of suffocation, particularly obstructions of the air passages. Physicians who are not discreet may unconsciously create in the child lasting and incapacitating fears of death. Often children are operated on without a word of preliminary explanation. Parents and others may even conspire to keep a child in ignorance, or even deceive it by untruths. The experience is usually neither forgotten nor forgiven. In cases of accident, children are quick to fear death. Without reassurance from physician or relative they may be left with an exaggerated fear of death, and this may for years frustrate them in many activities.

Chronic infirmities or disfigurements of any kind initiate insidious and deep-seated reactions. A child with such infirmities feels that he is different from other children. He fantasies himself marked off from them, and his life may actually become solitary. This frequently initiates feelings of inferiority. As the next step, the child begins to feel he has been punished unjustly and regards himself as the innocent victim of the world about him. His hostility is roused against all protectors such as God, and especially his parents.

Invalidism and self-pity may be responsible for undue selfishness, perhaps associated with self-assertiveness, in a child who has been pampered on account of ill health. Such children, when they grow up, share the fate of all egoists and suffer many a hurt to their feelings from a seemingly unkind world.

BECK, Buffalo.

HALLUCINATORY-CATAPLECTIC ANXIETY SYNDROME. C. ROSENTHAL, Arch. f. Psychiat. **102**:1 (April) 1934.

Rosenthal discusses the occurrence of the hallucinatory-cataplectic anxiety syndrome in its relationship to narcolepsy on the one hand and to catatonia on the other. His studies were based on nine cases, seven of narcolepsy, one of chronic encephalitis and one of migraine.

The syndrome consists of the following symptoms: attacks of loss of voluntary and expressive movements, with undisturbed consciousness; intensive hallucinations perceived as real and present in practically all of the sensory fields; an overpowering sense of anxiety, and a feeling of being influenced by others. Usually the syndrome develops in persons who have narcolepsy, and it seems to depend on states of dissociation in which the loosening of the psychomotor functional unity which occurs in narcolepsy is combined with the disintegrative process of sleep. It occurs practically always in relationship to sleep and when the person is lying flat on his back. Pathologically, it is probably dependent on disease of the highest vegetative centers in the diencephalon, and it usually occurs in persons who also show disturbances of the endocrine system. Rosenthal discusses the relationship of this pathologic state with those encountered in narcolepsy and the motor disturbances of catatonic schizophrenia.

MALAMUD, Iowa City.

THE SIGNIFICANCE OF SEXUAL ASSAULTS ON CHILDREN BELOW FOURTEEN YEARS OF AGE FOR THE DEVELOPMENT OF MENTAL DISEASES AND OF CHARACTER CHANGES. AUGUSTA RASMUSSEN, Acta psychiat. et neurol. **9**:351, 1934.

This study helps to invalidate the indiscriminate belief, widely held today, that sexual trauma received during childhood plays a decisive rôle in the development of mental diseases and of social maladjustment in adult life. Because the accounts and "confessions" made by mentally deficient, hysterical and neuropathic persons concerning their sexual experiences are never reliable, Rasmussen based her research on cases selected from court records (1902-1914) in which the victims were medically examined, the delinquents were convicted and penalty was imposed for sexual assaults on, and corruption of, minors. Fifty-four cases were thus selected in which the average age of the victim was from 9 to 13 years, and the ultimate fate in adult life was surveyed as to mental health and social adjustment. Forty-six of the victims were none the worse for the experience. Many of them at the time of survey were married and had children, and several were stolid and commendable members of the community. In eight cases the status of victims at the time of the survey was not satisfactory. Three of these had become psychotic, one was considered "hysterical," another was "very nervous" and three had become prostitutes, one of whom died of dementia paralytica. However, the rôle of the sexual trauma as a decisive factor in causing mental diseases and abnormalities of deportment in these eight persons, to say the least, was doubtful, because in most of them, independently of psychic sexual trauma, there existed evidence of a constitutional predisposition to mental imbalance. YAKOVLEV, Palmer, Mass.

Meninges and Blood Vessels

ACUTE ASEPTIC MENINGITIS. HENRY R. VIETS and JAMES W. WATTS, J. Nerv. & Ment. Dis. **80**:253 (Sept.) 1934.

In 1925 Wallgren first reported a series of cases of meningitis in which the onset was acute but relatively mild and there were headache, vomiting and moderate fever. Recovery occurred usually without residual paralysis after from three to six weeks. The cerebrospinal fluid showed a marked pleocytosis without polymorphonuclear cells, with a variation of from 150 to 750 cells per cubic millimeter. The sugar and chloride contents of the cerebrospinal fluid were normal more or less constantly during the course of the disease. This factor serves to differentiate this disease entity from tuberculous meningitis, in which an opposite condition prevails. The colloidal gold curves were all in the meningitic zone. Since Wallgren, others have reported similar cases. Eckstein in 1930 injected sterile cerebrospinal fluid from three patients into the cisterna magna of three monkeys. Two monkeys remained well, but the third animal showed fever, ataxia and paralysis in both hindlimbs and forelimbs, and the spinal fluid showed marked pleocytosis. Histologic examination showed a nonpurulent meningomyelitis of the cervical portion of the spinal cord. Eckstein considered the disease to be an abortive form of epidemic encephalitis.

Viets and Watts include in the article five cases which have previously been reported and reports of nine additional cases. They propose the term acute aseptic meningitis for the disease because of its acute onset and because the inflammation of the meninges is nonpurulent or lymphocytic. In one case, however, polymorphonuclear leukocytes were found in the cerebrospinal fluid from the fifth to the nineteenth day. In two of the thirty-five cases reported by Andersen and Wulff chronic encephalitis developed. The authors believe, however, that there is little ground for connecting the two diseases and that it is best to consider this disease an entity until it is proved to be otherwise. HART, Greenwich, Conn.

TUBERCULOSIS OF THE CENTRAL NERVOUS SYSTEM IN CHILDREN. AGNES R. MACGREGOR, H. J. R. KIRKPATRICK and W. S. CRAIG, Lancet **2**:18 (July 7) 1934.

In the course of an investigation of cases of tuberculous meningitis and glandular tuberculosis, three cases have been observed which indicate that active tuberculous infection of the central nervous system, associated with the presence of tubercle bacilli in the cerebrospinal fluid, does not necessarily proceed immediately to progressive and fatal tuberculous meningitis. Tubercle bacilli were isolated from the cerebrospinal fluid of these patients, and their subsequent history suggested that the lesion in the nervous system had been at least temporarily arrested.

In these cases there was an absence of the classic evidences of meningitis and extreme mildness and transience of such nervous symptoms and signs as existed. There was an absence in two of the three cases of any definite chemical or cytologic change in the cerebrospinal fluid. None of the three patients was, in fact, at the time of the attack, thought to be suffering from meningitis. The cases seem, therefore, to fall into a category somewhat different from that of previously reported cases in which there was recovery from tuberculous meningitis. It is the belief of the authors that the proved occurrence of cases such as these may throw new light on the problem of the pathogenesis of tuberculous meningitis.

In the light of the facts recorded it is necessary to recognize that any clinical evidence of meningeal irritation or cerebral lesion, no matter how slight and transient, in a patient in whom the tuberculin reaction is positive, may be an indication of tuberculous infection of the central nervous system even in the absence of clinically active tuberculosis elsewhere in the body and of any definite cytologic or chemical abnormality in the cerebrospinal fluid.

BECK, Buffalo.

CEREBROSPINAL MENINGITIS WITH INFECTION BY PSEUDOMENINGOCOCCI. DOPTER, ANDRIEU and CROSNIER, Ann. de méd. **36**:306 (NOV.) 1934.

Since von Lingelsheim published his report of sporadic cases of cerebrospinal meningitis produced by micro-organisms which resembled closely the meningococcus of Weichselbaum in their morphology and their staining reaction (1906), a large amount of literature has accumulated on this subject of the "pseudomeningococci." The demonstration of other diplococcic forms in the nasopharynx of healthy persons together with the meningococcus was considered as a proof that the first group was saprophytic only and did not play any rôle in the production of cerebrospinal meningitis (Micrococcus catarrhalis, Diplococcus siccus, Diplococcus pharyngeus-flavus I and III). But in more recent years it was found that "saprophytic" diplococci were able to produce not only infections of the upper respiratory tract but cerebrospinal meningitis and more malignant forms of generalized septicemia as well. It was concluded that they resemble meningococci in their pathogenicity, since the meningitis was superimposed on a preceding infection of the nasopharynx. Besides, the clinical pictures of disease produced by these two different groups of micro-organisms could hardly be differentiated. The pseudomeningococci were found chiefly in cases of cerebrospinal meningitis occurring in children, in whom the mortality rate was high. The problem became still more complicated by the fact that "pseudomeningococci" had been found in the cerebrospinal fluid of patients in whom meningitis had been produced by an entirely different factor. There was the possibility that the organisms were present merely as saprophytes or that they had induced a meningitis which was superimposed on the primary inflammatory reaction. Since antimeningococcus serum is ineffective in the treatment for meningitis produced by the "pseudomeningococci," the use of autovaccines should be the therapy of choice.

WEIL, Chicago.

CLINICAL AND ANATOMICOPATHOLOGIC STUDY OF SUPPURATIVE PHLEBITIS OF THE CAVERNOUS SINUS. H. ISMAÏL, Ann. d'ocul. **171**:814 (Oct.) 1934.

Ismaïl states that the term "thrombophlebitis" of the orbit and cavernous sinus is often used incorrectly to designate a serious disease which is not associated with thrombosis and which is really suppurative phlebitis. The majority of cases of septic obliteration of the cavernous sinus and orbital veins are due to suppurative phlebitis and not to thrombophlebitis. Ismaïl concludes that phlebitis of the orbital veins and cavernous sinus is a complication that may appear in the course of infections of the face and skull, such as furuncle, erysipelas, suppuration of dental origin and injuries. While Ismaïl does not deny the possibility of thrombophlebitis, the only lesion demonstrated was suppurative phlebitis. The late cases, in which thrombophlebitis is verified at necropsy, were not found. This study was made to illustrate the difference between thrombophlebitis and suppurative phlebitis. The prognosis of orbitocavernous phlebitis is extremely grave. From 5 to 7 per cent of patients with that condition are cured. It is possible that the cases in which cure was effected include some cases of thrombophlebitis. The differentiation of thrombophlebitis and suppurative phlebitis has not been proved definitely. Prophylactic treatment should be instituted. A knowledge of the possibility of venous complications should avert infections of the cephalic region, especially of the face. Treatment is symptomatic. Eagleton alone has attempted operation on the cavernous sinus; he reports three cures in twenty-four cases.

BERENS, New York.

Diseases of the Brain

TUMORS ARISING FROM THE SENSORY ROOT OF THE TRIGEMINAL NERVE IN THE POSTERIOR FOSSA. M. A. GLASER, Ann. Surg. **101**:146 (Jan.) 1935.

From the study of the case reported, together with that of three additional cases collected from the literature, it appears as if a clinical syndrome of perineurial fibroblastoma of the fifth nerve may be deduced. Pain referable to the trigeminal nerve is absent because the ganglion itself is not involved. Evidence of involvement of the trigeminal nerve is a consistent and early symptom and is characterized by diminution or absence of the corneal reflex and anesthesia over the distribution of the trigeminal nerve, particularly the opthalmic division. With encroachment on the cerebellum nerve signs became apparent. Further pressure on the medulla and on the pons results in symptoms referable to the cranial nerve and to the pyramidal tract. The condition is of long duration, with a late development of intracranial pressure. Periods of remission are characteristic in all these cases. The vestibular tests and audio-amplifier are helpful in ruling out tumors in other areas.

Tumors arising in the ganglion itself usually cause pain as well as anesthesia in the division of the trigeminal nerve. Signs referable to the cerebellum appear extremely late and occur only when there is extension of the tumor beneath the tentorium. Perineurial fibroblastoma of the eighth nerve may be readily differentiated from these tumors by loss of hearing and a history of tinnitus of long standing. Vestibular tests in the case of these tumors are pathognomonic. Meningiomas generally have a more progressive course and are not subject to remission. Gliomas of the pons and medulla are readily differentiated by the bilaterality of symptoms, and the definite vestibular findings indicate destruction rather than pressure. In cases of tumors of the cerebellum the sequence of events is that of symptoms referable to the cerebellum first and associated symptoms last; in addition, vestibular tests are of great value.

GRANT, Philadelphia.

MENINGIOMAS ATTACHED TO THE MESIAL PART OF THE SPHENOID RIDGE. CHARLES A. ELSBERG and CORNELIUS G. DYKE, Arch. Ophth. **12**:644 (Nov.) 1934.

The object of Elsberg and Dyke in this paper was to call attention to a characteristic symptom complex of small meningeal growths which arise from the most

mesial portion of the sphenoid ridge. These growths lie in close proximity to one optic nerve, and for a considerable period the disturbances they produce are due solely to pressure on the nerve near the optic foramen. As the authors say, the syndrome of primary atrophy of the optic nerve, with bitemporal defects of the fields of vision and progressive diminution of vision, is characteristic of tumors which compress the optic chiasm and the nerves. This syndrome is often the first and for a long time the only clinical evidence of a midline growth beneath the frontal lobes of the brain. The syndrome may be produced not only by lesions of the hypophysis but also by cysts or solid growths derived from the bucconeural pouch or infundibular stalk, by primary gliomas of the optic chiasm, by localized inflammatory processes in the leptomeninges and frequently by midline meningiomas attached to the basilar dura of the anterior or of the middle cranial fossa.

Early recognition of suprasellar meningiomas was due first to Cushing, and later to Cushing and Eisenhardt. The publication of an article by these two men in 1929 seemed to inaugurate a new era in diagnosis. They made the following statement: "Certain other meningiomas, which ophthalmologists should be quick to recognize, take their origin from the basilar leptomeninges in the vicinity of the pituitary fossa. Many of them have a distinctive symptomatology . . . others with equally unmistakable manifestations arise from the sphenoidal ridge lateral to the sella turcica and implicate the adjacent optic and oculomotor nerves; still others may actually arise from the meningeal expansions constituting the sheath of the optic nerve within the cavity of the orbit."

More recently, Alpers and Groff described a series of eight meningiomas, four of which were attached to the dura of the lesser wing of the sphenoid bone and four to the greater wing of the sphenoid. They stated: "The type of clinical syndrome to be expected, therefore, from tumors of the lesser sphenoid wing consists of a visual field cut, which is usually a complete homonymous hemianopia but which may be quadrantic, with localizing signs indicating involvement of the base of the brain, such as primary optic atrophy, a Kennedy syndrome, paralysis of the third nerve and unilateral impairment of the olfactory nerve. Other symptoms, such as motor paralysis of a central type (monoplegia), impairment of memory and pituitary disturbances may be present . . . the sella turcica may be normal or may show changes similar to those described in parasellar lesions . . . slight erosion of the dorsum and the anterior clinoid processes and unilateral deformation of the sella turcica in some instances. . . . Usually the tumors of the lesser wing are small and are rather closely confined to the vicinity of this wing. . . . Usually the tumors compress one or both optic nerves and the optic tract on one side when they are in contact with the lesser wing; they may likewise compress the olfactory nerve of one side, the oculomotor nerve, the uncus and the internal carotid, the middle cerebral or the anterior cerebral vessels."

The combination of symptoms described by the authors quoted is characteristic of large growths derived from the dura of either the lesser or the greater wing of the sphenoid, and many of the subjective and objective disturbances are due largely to the large size of the neoplasm and to involvement of many areas of the brain. Tragically, not a few patients are admitted to the hospital and subjected to operation when a neoplasm has reached such a size that it may weigh from 50 to 200 Gm. or more. A better appreciation by the patient of the importance of slight visual disturbances and by the physician of the significance of slight symptoms is needed.

Among the favorite sites of attachment for meningiomas at the base of the brain are the dural covering of: (1) the tuberculum sellae, (2) the olfactory groove and cribriform plate, (3) the roof of the orbit, (4) the superior surface of the lesser wing of the sphenoid, (5) the outer part of the sphenoid ridge and (6) the mesial part of the sphenoid ridge. When they are small the meningiomas attached to the dura at the sites mentioned under 3, 4 and 5 do not come into contact with structures that have a specialized function; therefore, it is not surprising that for a long time many of the patients are free from disturbances which would lead them to consult a physician.

Further, a small growth attached to the dura of the olfactory groove must cause unilateral disturbances of smell very early. The value of encephalography for patients with disturbances of smell not clearly due to intranasal disease is self-evident.

The authors present four cases of tumor attached to the most mesial part of the sphenoid ridge. The syndrome of unilateral atrophy of the optic nerve, with a defect of the visual field on the same side, with very slight roentgenologic changes of the sella turcica and with alterations in the shape of the interpeduncular cistern is characteristic of tumors in this situation. In each instance the main complaint was a slowly progressive diminution of vision in one eye. Other symptoms, which usually occur in intracranial growths in this region, were absent. In each instance the neoplasm was attached to the sphenoid ridge near one anterior clinoid process, so that from the beginning the optic nerve was subjected to pressure. In all cases slight atrophy of the floor of the sella or of one clinoid process was found by the roentgenologist, but in each instance the exact situation of the neoplasm was determined only by study of the roentgenogram after the lumbar injection of air.

SPAETH, Philadelphia.

EPENDYMOMA OF THIRD VENTRICLE. A. R. VONDERAHE and N. R. ABRAMS, Arch. Ophth. **12**:693 (Nov.) 1934.

Involvement of the optic chiasm from its inferior surface occurs with fair frequency owing to the high incidence of neoplasms of the pituitary gland. Involvement of the optic chiasm from its superior surface, that is, from impingement on it by a lesion proceeding from the floor of the third ventricle, occurs less frequently, and actual invasion of the chiasm is extremely rare. Vonderahe and Abrams report such a case. It was one of direct invasion of the optic chiasm by an ependymoma of the third ventricle. There were various vegetative manifestations, increased intake and output of fluids, sexual impotence, increased pigmentation of the skin, lethargy and premature graying of the hair followed by baldness. An explanation of these findings is found in involvement either by pressure or by invasion of the vegetative area of the brain, implicating chiefly the tuber cinereum and the floor and walls of the third ventricle.

Wilbrand and Saenger first pointed out that direct invasion of the chiasm by a tumor is extremely rare. Several others have reported such instances, but they are uncommon.

The authors' patient was operated on by a subtentorial craniotomy. No tumor was found. Fifteen days later a further exploratory craniotomy was done in the left frontotemporal region. Thirteen days later death occurred. Postmortem examination showed a tumor obliterating the cavity of the third ventricle, with hemorrhages into the mass, cerebral edema and congestion and internal hydrocephalus of both lateral ventricles secondary to an obstruction of the aqueduct of Sylvius. Microscopically, there were other widespread pathologic changes. The nerve fiber pathways in the corpus callosum, immediately superior to the third ventricle, presented marked degenerative changes, evidently an effect of pressure. The columns of the fornix in the roof of the third ventricle were similarly affected. There was marked cell loss in the thalamus. The fiber tracts of the internal capsule on each side showed loss of myelin. The anterior commissure was invaded by the tumor. The globus pallidus showed a loss of cells. The optic chiasm was directly invaded by the tumor. The tuber cinereum was completely replaced by neoplastic growth.

SPAETH, Philadelphia.

UNILATERAL OPHTHALMOPLEGIA TOTALIS; PARASELLAR OSTEOCHONDROMA. JESSE M. LEVITT, Arch. Ophth. **12**:877 (Dec.) 1934.

Rollet classified tumors in the region of the sella turcica into four groups with respect to ocular signs: (1) the optic or amaurotic form, in which the signs are the result of compression of the visual fibers and which includes the familiar

chiasmatic syndrome and bitemporal hemianopia; (2) the sensorimotor and cavernous or vascular form, in which the signs are the result of pressure on the trunks of the ocular nerves in the wall of the cavernous sinus and on the cavernous sinus itself; (3) the total or opticocavernous form, combining signs of the first two forms, and (4) the exophthalmic form, in which the predominant sign is proptosis. Levitt presents a case which corresponds to the second type in the groups mentioned. He thinks that it is a rare clinical entity. In reviewing the literature of the past thirty-three years he found no additional cases.

Rollet, in discussing the causes of tumors in group 2, distinguished two subtypes: a pure sensorimotor type characterized by multiple paralysis of the ocular nerve and ophthalmoplegia, and a sensorimotor and vascular type, combining vascular changes such as chemosis with signs of involvement of the nerve.

Levitt's case was that of a woman, aged 20, with ptosis and blurred vision of the left eye. There was a total external ophthalmoplegia on the left side. The fundi were normal. The fields were normal. There was unilateral exophthalmos of slightly less than 2 mm. There was an almost complete internal ophthalmoplegia as well. Roentgenographic studies revealed irregular calcific deposits above and behind the sella turcica and extending slightly therein. Operation showed a small nodular grayish-yellow tumor on the sphenoid ridge at its junction with the left anterior clinoid process. Microscopic sections of fragments of the tumor showed the presence of fairly well developed bone surrounded by periosteal tissue and containing islands of bone marrow. In addition to these there were islands of embryonal cartilage cells, simulating myxoid tissue. The diagnosis rested between osteochondrosarcoma and osteochondromyxoma.

The patient had an uneventful convalescence. Nine weeks after the operation the upper lid was in a normal position. There were absence of the winking reflex, almost complete restoration of the function of the internal rectus muscle and partial restoration of that of the external rectus muscle and complete residual internal ophthalmoplegia.

This case illustrates the early type of basal intracranial tumor with a pure clinical syndrome of the external wall of the cavernous sinus.

SPAETH, Philadelphia.

A CASE OF CEREBRAL METASTATIC MELANOMA SIMULATING CEREBROSPINAL MENINGITIS AND ENCEPHALITIS. J. GREWAL and W. KELLY, Psychiatric Quart. **8**:276 (April) 1934.

A case of a cerebral melanoma producing symptoms suggestive of encephalitis or meningitis is presented. The patient, a man, had had a pigmented birth mark on the right abdominal wall which was cauterized and removed when he was 8 years of age. Resection had apparently been effective, as no local recurrence was ever noted. At the age of 20, the patient began to lose weight and strength. This loss was gradual and scarcely noticed. When he was 26 there developed a "cold" with generalized pains and aches. Within a few weeks thereafter mental symptoms appeared. The patient became irritable, quarrelsome, garrulous, delirious, disoriented and noisy. The muscles of the back and neck became stiff, attacks of vomiting occurred, the pupils were dilated, the right pupil being larger than the left, and an internal strabismus was noted. The neck was overextended and the knees were flexed. Ophthalmoscopy showed bilateral papilledema, more marked on the left. The Kernig sign was positive, and the leukocyte count was 12,700, 36 per cent of the cells being polymorphonuclears. The spinal fluid was cloudy; the pressure, 14 mm. of mercury, and the cell count, 800, the forms being mostly polymorphonuclears. The globulin content of the spinal fluid was increased, and the colloidal gold curve was 0000012333. No organisms were found in the fluid. The patient died three months after the onset of the "cold."

At autopsy the dura mater was found to be thickened and adherent. The pia was cloudy. The cerebral vessels were congested. Over many areas of the cortex there extended a dense subpial sheet of brown pigmentation. The meninges within

the sylvian fissure were laden with pigment. Two large tumor masses were present in the frontal lobe and extended into the precentral gyrus. They were dark brown. Similar tumor masses were found in the spleen, liver and adrenal glands. Histologic study revealed brown-black pigment within the cytoplasm of the tumor cells. The perivascular spaces of the brain were completely filled with the pigmented tumor cells. The authors believe that the original melanoma was the pigmented birth mark on the abdominal wall, from which site it metastasized to the adrenal glands, liver, spleen, retroperitoneal lymph nodes, meninges and brain.

DAVIDSON, Newark, N. J.

THE GENERALIZED AND VERTEBRAL FORMS OF MYELOMA: THEIR CEREBRAL AND SPINAL COMPLICATIONS. P. G. DENKER and S. BROCK, Brain **57**:291, 1934.

Myeloma may be regarded as a malignant tumor of the bone marrow arising either from a single cell type, the plasma cell, or perhaps from a number of different bone marrow elements. There is still much difference of opinion concerning the pathogenesis of these growths. The plasma cells or the myeloblasts of the bone marrow are regarded by many observers as the original cells. Others believe the type cell to be a transitional form, neither plasma cell or myelocyte. The osteoblast has been considered. Witzleben (1925) regarded myeloma as a systemic disease of the blood-forming organs, closely allied to the leukemias, but aleukemic in nature, with predominant participation of the bone marrow and occasional involvement of other hematopoietic tissue. Lubarsch (1906) believed that myeloma belongs to the group of hyperplastic diseases and not to that of truly neoplastic diseases.

The disease is rare. The short flat bones are especially involved. Soft gray or reddish-gray masses start in the marrow, grow outward and replace the cortex of the bones. Secondary growths may appear in the liver, spleen, kidneys, lungs and sex glands. The disease occurs in the later period of life and is seen twice as often in men as in women. In no other type of tumor of the bone does pathologic fracture occur so frequently. Bence-Jones bodies are found in the urine in a majority of the cases. An increase in protein content of the plasma and large amounts of amyloid deposits have been reported. Nephrosis, anemia and a chronic low grade fever have been reported in many cases. The prognosis is grave, the average duration of life being two years.

Denker and Brock report one case of the generalized form of myeloma and three cases of the vertebral form. The generalized form presented some unusual features. The patient, a girl, aged 15, suffered from recurrent convulsive seizures and polydypsia. Roentgenograms showed widespread destruction of bone throughout the skeleton, with marked involvement of the skull. At autopsy there were deposits of lime in the kidney and atrophy and fibrosis of the pancreas, with concretions of the ducts. The convulsions were attributed to the cerebral effects of the countless bony tumors in the skull. The polydypsia was regarded as part of a diabetes insipidus due to bony invasion of the basilar parts of the skull with secondary pressure on the pituitary gland or on the structures of the floor of the third ventricle.

Isolated forms of myeloma have been considered very rare, and some authors doubt the existence of such forms. Recent reports and the experience of Denker and Brock show that the isolated vertebral form is not so rare. The clinical separation of myeloma into the vertebral form seems justified, because for a considerable period of time the growth may be confined to the vertebral and extradural tissues and the patient may die before the growth becomes widespread. In the vertebral form the typical picture presented is that of a transverse spinal lesion with manometric block. The upper part of the thoracic portion of the spinal cord is usually the site of compression. There is a local area of tenderness. Roentgenograms of the spine may show normal conditions, or there may be destruction of the vertebral body. No primary focus of malignant degeneration can be demonstrated elsewhere.

Three cases of the vertebral form are reported. In all, the signs pointed to compression of the cord with manometric block. Roentgenograms showed no bony change in the vertebrae in one case and evidence of destruction of a vertebral body in two cases. Two patients died, and autopsy showed the myelomatous process to be relatively circumscribed to the vertebrae and adjacent ribs. Two patients were operated on; one patient is still alive and shows great clinical improvement nine months after operation. Reexamination and roentgenographic studies show no new myelomatous foci. In the other patient marked clinical improvement occurred for over two years after operation, then the patient presented evidence of a recurrence in the same location in the thoracic portion of the cord. She was again operated on, and again there was clinical improvement for six months, then signs of a transverse lesion of the cord suddenly appeared. The patient died after the development of an infection of the bladder. Roentgenograms of the skull and skeleton had given repeatedly negative results in this case. In all the cases microscopic study of tissue removed at operation and at autopsy revealed features characteristic of myeloma.

SALL, Philadelphia.

BASOPHIL ADENOMA OF THE PITUITARY GLAND. DOROTHY S. RUSSELL, HORACE EVANS and A. C. CROOKE, Lancet **2**:240 (Aug. 4) 1934.

The clinical and pathologic features in two proved cases of basophil adenoma of the pituitary gland are described. The first is noteworthy in that it is not only the second verified case in a male patient but also in that it presented severe atypical features. The second case, that of a female patient, is of importance because only a few of the characteristic features of the syndrome were present. The salient feature in both patients was cardiovascular hypertrophy. Obesity was present in both, but in one it was atypical. Deposition of fat was general, affecting the limbs. One of the patients died of nephritis; the other died of cerebral hemorrhage complicated by essential hypertension and primary ischemic nephritis. In neither case was there an abnormal invasion of the posterior lobe by basophil cells; this ruled out Cushing's recent suggestion that basophilic invasion of the posterior lobe might be potent in producing the outstanding features of this illness.

BECK, Buffalo.

CYSTICERCUS CYST OF THE THIRD VENTRICLE. J. A. BARRÉ, Ann. de méd. **36**:275 (Nov.) 1934.

The main clinical features in this case were mildly increased intracranial pressure, mild hemiparesis on the right side with mild hypertonicity and normal tendon reflexes, disturbances of the vestibular apparatus, sluggishness of the pupillary reactions to light and in accommodation and inability to converge the left eye. There were secondary atrophy of the optic nerve and nasal hemianopia in the right eye and papilledema in the left eye. Toward the end of the disease there appeared somnolence, varying in intensity during different periods, and paralysis on the left side. Autopsy revealed a multilocular Cysticercus cyst which completely filled the third ventricle. Smaller cysts were found in the temporal lobe and within the pia-arachnoid at the base of the brain. Here they had led to an intense leptomeningitis with a dense leukocytic infiltration.

WEIL, Chicago.

MULTIPLE METASTASES OF A PAPILLOMA OF THE THYROID GLAND IN THE CENTRAL NERVOUS SYSTEM. M. ZAIEWLOCHINE, Encéphale **29**:609 (Nov.) 1934.

The case of a woman aged 48 is reported. There were many neurologic signs. At autopsy primary carcinoma of the thyroid was found. This had metastasized into the lymphatics of the neck, into the right cerebral hemisphere and into the cranial bones. There was also softening of the thoracic segments of the spinal cord. Such tumors are very rare, only thirty-one cases having been reported up to 1927.

ANDERSON, Los Angeles.

ISOLATED ABSCESS OF THE BULB. EGAS MONIZ, Rev. d'oto-neuro-opht. **12**:568 (Sept.-Oct.) 1934.

Abscess of the bulb, and especially isolated abscess, is extremely rare. The patient whose case is reported entered the hospital in February 1931 suffering from continual hiccup, dysarthria, vomiting and difficulty in masticating and swallowing. In 1930 he had an attack of pleurisy accompanied by suppuration of the right ear. For six months before admission he suffered from neuralgia of the right side of the face. Partial examination showed diminished vision in the right eye, falling to the left and facial paresis on the right side. Death occurred suddenly in the first twenty-four hours. Autopsy revealed an isolated abscess in the right posterior part of the bulb. The most common bulbar lesions occur by way of the blood vessels. Moniz believes that the source of the infection was suppuration of the mastoid, though the influence of the pleurisy cannot be eliminated.

DENNIS, Colorado Springs, Colo.

TUMORS OF THE SELLAR REGION. D. MANOLESCO and G. SCHMITZER, Rev. d'oto-neuro-opht. **12**:661 (Nov.) 1934.

From a study of a large number of patients with tumors of the sellar region the following conclusions are drawn: 1. In order to establish the existence of hemianopia examination of the visual fields for colors is indispensable, because hemiachromatopsia precedes hemianopia. 2. For the diagnosis of hemianopia in cases in which the visual acuity is nil, examination for luminous perception in the different zones of the visual field is necessary. 3. In cases of paralysis of the oculomotor nerves, examination of the visual fields furnishes important information. 4. Roentgenotherapy should be used in all cases of tumor of the sellar region without regard to their nature. Surgical intervention is indicated only in cases in which the roentgen rays have not been beneficial. 5. The return of the visual fields after roentgenotherapy occurs separately for each color by islands, which coalesce.

Six cases are reported. Roentgenotherapy was beneficial in all.

DENNIS, San Diego, Calif.

PARALYSIS OF OCULOGYRIC MOVEMENTS IN MULTIPLE SCLEROSIS. H. ROGER, J. ALLIEZ and J. PAILLAS, Rev. d'oto-neuro-opht. **12**:747 (Dec.) 1934.

Since the work of Barré, the authors have discovered vertiginous episodes, which have at times preceded by months or years the apparent beginning of cases of multiple sclerosis. They report the case of a young man with oto-ophthalmologic symptoms; he experienced two evolutive episodes, separated by an interval of eighteen months of complete good health, each characterized by predominance of the ocular and equilibrial disturbances: paresis of the left sixth nerve in both attacks and paralysis of levogyric movements in the first attack and of the dextrogyric movements in the second. A diagnosis of multiple sclerosis, with the chief foci in the left peduncular region, appeared to be the only one possible. The paralysis was total, which is exceptional. Diplopia and horizontal nystagmus to the left and affecting chiefly the left eye were noted.

In these cases vertical paralyses are rare; paralysis of lateral movements occurs in 20 per cent of the cases. In some cases various associated movements are simultaneously affected. In the case of Raymond and Cestan there was paralysis of elevation, laterality and convergence. Autopsy revealed an enormous plaque in the pedunculopontile region, embracing the nuclei of the third and fourth nerves and the anterior part of the corpora quadrigemina. These paralyses of function are to be attributed to disturbances of the vestibular pathways. In the case reported by Roger, Alliez and Paillas, there were no nystagmus responses to caloric or turning tests, but there were past pointing and a violent swaying to the right. Most authorities attribute this symptom to an irritative or a destructive lesion of the posterior longitudinal bundle.

DENNIS, San Diego, Calif.

CUTANEOCEREBRAL ANGIOMA. J. S. CHARAMIS, Rev. d'oto-neuro-opht. **12**:755 (Dec.) 1934.

Certain cutaneous vascular lesions, such as facial vascular nevi, may be accompanied by true cerebral angiomas. Often they are accompanied by angioma of the retina. These cases must not be confused with those of angiogliomatosis of the retina, atypical cases of Recklinghausen's disease with cerebral lesions, or with pure cerebral angioma without cutaneous or ocular lesions. A boy, aged 14, presented mucocutaneous angiomatous nevi in the region supplied by the right trigeminal nerve, with a homolateral cerebral angioma; there were no signs of a neurologic pathologic process except epileptic attacks and mental dulness, but pronounced calcification of the vascular tumor, syphilitic iridocyclitis in the right eye, and positive reactions to the Wassermann and Hecht tests were present. While the pathogenesis of cases of this type is not settled. Charamis is inclined to attribute the cause of the diseases in this case to syphilis. Active antisyphilitic treatment and high voltage roentgen therapy were applied. If the cerebral complications become menacing, extirpation of the tumor is indicated, in spite of the attendant risks.

DENNIS, San Diego, Calif.

Vegetative and Endocrine Systems

THE WATER AND SOLID CONTENT OF THE BRAINS OF ALBINO RATS TREATED WITH GROWTH HORMONE. H. S. RUBINSTEIN and L. M. FOX, J. Comp. Neurol. **60**:349 (Oct.) 1934.

To study the water and solid content of the brains of albino rats which have been given injections of a growth hormone, thirteen males were used. Five received the hormone; the others received injections of meat extract or were not given injections. All controls were litter mates of the experimental animals. Injections were begun at 32 weeks of age and were given intraperitoneally six times weekly for twenty-two weeks. The average gain in body weight of the animals given injections of the growth hormone was 104 Gm. as compared with 46 and 39 Gm. for the animals given injections of meat extract and for the untreated animals, respectively. The mean weight of the brain of the experimental animals was lighter than that of the control groups. The weight of the dried brain was proportionally smaller in the test animals than in the controls. The percentage of water was the same for all groups. The water and solid contents of the brains of the animals given injections remained within the normal limits. The authors conclude that the growth hormone fails to increase the size of the brain in proportion to the body.

ADDISON, Philadelphia.

ACROMEGALY IN ONE OF UNIOVULAR TWINS. A. LEWIS, J. Neurol. & Psychopath. **15**:1 (July) 1934.

The only indisputable case of acromegaly recorded in one of a pair of uniovular twins, proved by the history and by footprints and fingerprints, is reported. The patient, a man aged 46, in good health, began to show developmental differences from his brother early in childhood; he grew 8½ inches (21.6 cm.) taller, besides exhibiting typical acromegaloid features. The twins were affected by similar illnesses at the same time in their earlier years. The acromegaloid twin, however, experienced trauma to the head at the age of 12, to which no great significance is ascribed. The rarity of the occurrence of acromegaly in families is suggestive of the unimportant part that heredity plays in this condition, although there is an inherited predisposition for dysfunction of the anterior lobe of the pituitary gland. The occurrence of many tall persons in the family tree in the case reported is suggestive of this fact.

SPERLING, Philadelphia.

AN ADRENALINE-LIKE ACTION IN EXTRACTS FROM THE PROSTATIC AND RELATED GLANDS. U. S. VON EULEN, J. Physiol. **81**:102 (March 29) 1934.

Using watery alcoholic extracts from the prostate glands of man and the dog, rabbit and guinea-pig, from the vesicular gland of the steer and sheep and from the ampulla ductus deferentis of the dog, steer and sheep, von Eulen has observed pressor action and inhibitory action on the isolated intestine of the rabbit. Inactivation experiments, the presence of the Vulpian iodine reaction in the extracts and the finding of chromaffin cells within the muscular wall of the ampulla ductus deferentis and the prostate gland, as well as characteristic changes in action after the administration of cocaine and ergotamine, indicate that the actions observed are due to epinephrine. The amount of the active substance corresponds to from 0.001 to about 0.005 mg. of epinephrine per gram of fresh organ.

The presence of such amounts of an epinephrine-like substance as have been found in the extracts referred to in this paper seems to indicate a special function of these organs as epinephrine-producing. Opinions have differed as to the question whether the prostate gland has a function as an endocrine organ. Most investigators and reviewers have taken the standpoint that sufficient evidence has not been produced to allow such a theory. By the demonstration of a substance probably identical with epinephrine in comparatively large amounts in this organ, the assumption of an endocrine function is rendered more likely. The possible function of some of the male accessory genital glands as endocrine organs might then be regarded in connection with the physiology of these organs, especially in view of the fact that epinephrine, like stimulation of the sympathetic nerves in this region, will cause contraction of the plain muscle in general of the genital apparatus.

ALPERS, Philadelphia.

STUDIES ON THE PHYSIOLOGY OF THE THYREOTROPIC HORMONE OF THE ANTERIOR PITUITARY. EVELYN M. ANDERSON and J. B. COLLIP, J. Physiol. **82**:11 (Aug. 24) 1934.

The close relationship between the anterior lobe of the pituitary gland and the thyroid gland has been clearly demonstrated both experimentally and clinically. Hyperplasia of the thyroid gland has been produced by injections of extracts of the anterior lobe of the pituitary gland in a number of laboratory animals, such as the guinea-pig, cat, dog, rabbit, pigeon and duck. Anderson and Collip were able to produce severe hyperplasia of the thyroid gland with hyperthyroidism in the rat, using crude extracts of the anterior lobe of the pituitary gland combined with killed cultures of staphylococcus vaccine. Since this work they have been able to obtain extracts with thyreotropic hormones of sufficiently high potency to cause hyperthyroidism in the rat without simultaneous use of staphylococcus vaccine. Others have shown that none of the signs of hyperthyroidism can be produced in thyroidectomized animals after the injection of extracts of the anterior lobe of the pituitary gland. Anderson and Collip used rats and young guinea-pigs in their experiments. Extracts of the anterior lobe of the pituitary gland of oxen and sheep containing the thyreotropic hormones were used, and their effect was compared with that of extracts of the anterior lobe of the pituitary gland free from thyreotropic hormone, extract containing growth hormone, and extract containing the anterior pituitary-like principle of the urine of pregnant women; normal controls were also observed. Hyperthyroidism as indicated by an elevated metabolic rate was produced by purified extract of the hormone. The thyroid glands of treated animals were more than twice the weight of those of the normal controls and histologically showed severe hyperplasia such as is seen in the thyroid gland in cases of exophthalmic goiter.

The earlier work, in which the authors induced hyperthyroidism in rats by means of staphylococcus vaccine used in conjunction with crude extracts of the anterior lobe of the pituitary gland, was repeated, purified extracts of the hormone being used. The findings indicate that the staphylococcus vaccine increases the sensitivity to the thyreotropic hormone.

After hypophysectomy, atrophy of the thyroid gland occurs and is associated with a decrease in metabolism. The average metabolic rate of 118 hypophysectomized rats was 74 per cent of the normal. The rate may be raised temporarily to a normal level by administration of the purified thyreotropic extract in amounts as low as 0.02 cc. a day. The effect of prolonged treatment of the normal rat with large doses of thyreotropic hormone is a final lowering of the metabolic rate to the level obtained in the hypophysectomized rat owing to the production of an antithyreotropic substance. There is evidence that this substance is not formed by the pituitary gland, since in the hypophysectomized rat there develops a similar resistance to the hormone. A group of rats with goiter of unknown etiology, in which the metabolic rate was slightly below normal, were given thyreotropic hormone. Severe hyperthyroidism was produced; it was associated with an increase in the metabolic rate, the rate being as high as 262 per cent of normal in one instance. The method of preparation of a highly purified extract of the thyreotropic hormone is described by the authors. This preparation has been found to be stable if kept in the refrigerator in sterile ampules. It is not active when administered by mouth, and it is destroyed by prolonged boiling. PALMER, Philadelphia.

FURTHER EXPERIMENTS ON THE RELATION OF THE PITUITARY GLAND TO THE ACTION OF ADRENALIN AND INSULIN. O. COPE and H. P. MARKS, J. Physiol. **83**:157 (Dec. 31) 1934.

Some of the most striking consequences of total removal of the pituitary gland in the dog are the marked increase in sensitivity to insulin and the tendency to spontaneous hypoglycemia. This has been described by many workers. Marks and his co-workers confirmed these observations on rabbits. The present paper, in addition to presenting a fuller discussion of this mechanism, extends the previous investigation, particularly with regard to the possible relation between the increased sensitivity to insulin and the degenerative changes occurring in the thyroid gland. In this connection the authors studied: (1) the increase in the response to insulin produced first by thyroidectomy and second by subsequent hypophysectomy; (2) the effect of administering extract of the anterior lobe of the pituitary gland to thyroidectomized rabbits, and (3) the effect on the response to insulin of the administration of thyroid hormone to hypophysectomized rabbits.

Since the adrenal cortex undergoes extreme degeneration in cases of pituitary deficiency, the authors attempted to relieve this deficiency by the administration of cortical extract. Rabbits were used for all experiments, and complete hypophysectomy was performed. All injections of insulin were given intravenously. Epinephrine was injected subcutaneously. After removal of the pituitary body, the adrenal glands respond normally to the hypoglycemia that follows the injection of insulin by liberating epinephrine into the blood stream, yet this epinephrine fails to restore the lowered blood sugar content to the normal level, in spite of the presence of ample reserves of glycogen in the liver. The hyperglycemic response to injected epinephrine is likewise diminished. Converse changes, resistance to insulin and an increased response to epinephrine, are produced by injection of a suitable extract of the anterior lobe of the pituitary gland. The changes mentioned are explained on the supposition that the anterior lobe of the pituitary gland contains a principle which renders the reserves of glycogen in the liver and possibly also those in the muscles susceptible to the mobilizing action of epinephrine. The changes following removal of the pituitary glands cannot be reversed by the administration of thyroid; hence they are not due to concurrent degeneration of the thyroid. Conversely, the changes produced by injection of extract of the anterior lobe of the pituitary gland are not prevented by removal of the thyroid glands.

It is of interest to note that the mobilization of body fat normally brought about by the administration of thyroid does not appear to occur in the absence of the pituitary gland. This lends support to Houssay's view that the pituitary gland plays an essential part in the utilization of fat in the organism. With regard to the rôle of the adrenal cortex, Cope and Marks have little to offer. As a result

of one experiment they only tentatively suggest that the hypersensitivity to insulin of the hypophysectomized animal is unaffected by the administration of cortical extract. The cevitamic acid content of the cortex does not appear to be influenced by function of the pituitary gland.

PALMER, Philadelphia.

THE SPECIAL POSITION OF THE SACRAL PARASYMPATHETIC SYSTEM IN THE RADIATION OF AUTONOMIC REFLEXES. A. SCHWEITZER, Arch. f. d. ges. Physiol. **235**:110, 1934.

Impulses in the somatic part of the nervous system (acoustic and tactile stimuli) induced an increase of tonicity and contractility of the urinary bladder, while a reflex action of the carotid sinus on the bladder was absent. The sacral portion of the parasympathetic system is closely connected with the somatic system. Impulses from the carotid sinus radiate to the sacral parasympathetic system only after depressing the state of excitation of the somatic system; yet such impulses from the carotid sinus may act on respiration and on the stomach without changing the state of excitation of the somatic system. Increase of tonicity in the cranial part of the autonomic system (vagus) may be associated with an increased tonus of the sympathetic system as far as the bladder is concerned, as shown by the action of morphine. Epinephrine, while stimulating the sympathetic system in general, induces contraction of the detrusor muscle of the bladder which is supplied by the parasympathetic system. Schweitzer observed that after transverse section of the midbrain, impulses from the carotid sinus radiated only to the respiratory center and no longer stimulated the stomach and urinary bladder. It is supposed that autonomic reflexes radiate in the hypothalamic nuclei.

E. SPIEGEL, Philadelphia.

Treatment, Neurosurgery

THE ATROPINE TREATMENT OF THE POST-ENCEPHALITIC PARKINSONIAN SYNDROME. FELIX ADAMS and POWELL HAYS, Am. J. Psychiat. **91**:151 (July) 1934.

Excellent results are reported in the treatment of postencephalitic paralysis agitans by atropine. In the original regimen, toxic symptoms (nausea, vomiting and incontinence) developed in most of the patients but vanished when the atropine was discontinued. However, Adams and Hays have devised a new regimen of doses which has substantially abolished these toxic symptoms. Their program consists of the administration of a 0.5 per cent solution of atropine sulfate. The initial dose is 1 minim (0.06 cc.) three times a day. The dose is increased by 1 minim for the three daily doses each day until the fourth day. For three days the dose is maintained at 4 minims (0.24 cc.); then it is increased by 1 minim every day until it reaches 8 minims (0.5 cc.); this is maintained for three days, and on the thirteenth day the increase is resumed, the first dose each day being 1 minim greater than on the day before, until the optimum dose is established. The optimum dose commonly falls between 12 and 18 minims (0.74 and 1.12 cc.) three times a day.

The results of treatment of seventy patients are reported. In 90 per cent the physical condition was improved. Rigidity was improved in half of the patients, and in an additional third it was entirely eliminated. Tremor was improved in a third of the patients, unimproved in a sixth and practically abolished in one-half. Disturbances of gait and speech were improved in 95 per cent of the patients, and oculogyric crises were almost always controlled. After the optimal dose is established the patients should be fitted with glasses to overcome the mydriasis. The atropine should be continued indefinitely.

DAVIDSON, Newark, N. J.

THE MECHANICAL MANAGEMENT OF BRAIN ABSCESS. A. W. ADSON and W. McK. CRAIG, Ann. Surg. **101**:7 (Jan.) 1935.

Adson and Craig review 97 cases of abscess of the brain. Cases of epidural abscess were excluded. Sixty of the patients were male and thirty-seven, female.

The average age was 24. Three patients were less than 3 months of age. Forty abscesses were situated in the frontal lobe, 36 in the temporosphenoid area, 2 in the occipital area and 13 in the cerebellar area. In the remaining 6 cases the abscesses were distributed in other scattered areas. The invading organisms were Staphylococcus albus or Staph. aureus in 19 cases and Streptococcus haemolyticus in 11 cases. Headache was present in 79 cases, nausea and vomiting in 61, choked disks in 49 and blindness in 10. Fifteen patients had preoperative jacksonian convulsions and 20 had grand mal seizures. The neurologic symptoms varied according to the size and position of the abscess. The degree of recovery was in relation to the injury done by inflammation and surgical manipulation.

With regard to mortality, the results improved with the experience of the surgeon. The mortality rate dropped as soon as the surgeon delayed drainage until encapsulation took place. In cases of abscess of the temporosphenoid lobes there was a lower mortality rate when drainage was established through a clean field in the temporal area than when it was carried out through a suppurating mastoid wound. The sealing of the meninges and cortex about the drainage tract to prevent contamination has improved convalescence and lowered the mortality rate. Adequate continuous drainage with gauze packs and rubber tube drains has prevented recurrent abscesses and cerebral herniation. By these means, 14 of 15 consecutive cases of abscesses situated above the tentorium and 2 of 3 cases of cerebellar abscesses recovered.

Adson and Craig think that it is well to administer supportive treatment during the initial stage of infection and to delay drainage until clinical signs indicate that immunity has been established. They also prefer the instituting of adequate continuous drainage through a surgically sealed wound to the various conservative or ultraradical measures employed.

GRANT, Philadelphia.

BRAIN ABSCESS: EXTERNAL RUPTURE OF "CAPSULE" WITH PERICAPSULAR BRAIN NECROSIS. J. E. J. KING, Ann. Surg. **101**:190 (Jan.) 1935.

A case of abscess of the brain is reported, with details of a successful procedure whereby a collapsed wall of the abscess, or "capsule," was extirpated, together with the surrounding necrotic, purulent brain tissue. The suggestion is made that this procedure may some day be utilized in certain suitable cases of suppurative encephalitis or "phlegmon" of the brain, in which there is but little tendency for the abscess to encapsulate and in which nature does not seem to be able to limit the infection. When possible, an abscess of the brain should be dealt with in such a way as to remove it entirely by resorting to herniation rather than by mere drainage, which frequently leaves a residual septic abscess focus.

GRANT, Philadelphia.

THE SELECTION OF PITUITARY CASES FOR SURGERY. J. M. MEREDITH, Ann. Surg. **101**:228 (Jan.) 1935.

As is amply demonstrated by a review of the literature, there is great diversity of opinion concerning the indications for operation in cases of adenoma of the pituitary gland. There exists a diversity of opinion as to when to employ irradiation and when to employ surgical measures. It seems wise to limit roentgen therapy to cases in which there is little or no visual impairment, to cases in which there is headache only or to cases in which there are symptoms of disturbance of the endocrine system and no visual involvement. All patients subjected to irradiation before or in lieu of operation should be temporarily hospitalized and not treated as ambulatory patients, so as to combat any acute catastrophe caused by the treatment and to make possible immediate operation if necessary to conserve vision and even life. The case here reported demonstrates that although it is the consensus that chromophil adenomas are more susceptible to irradiation than is adenoma of the chromophobe type this is not always true; furthermore, that an excellent result may be obtained by operation after the failure of prolonged irradiation.

GRANT, Philadelphia.

SPINA BIFIDA AND CRANIUM BIFIDUM. S. W. GROSS and E. SACHS, Arch. Surg. **28**:874 (May) 1934.

This report is based on a study of ninety-eight cases of spina bifida, including two cases in adults and five cases of cranium bifidum observed in one clinic in twenty years. The indications for operation were the presence of some function in the lower extremities and the absence of pronounced hydrocephalus, of ulcerated or infected sacs and of extensive defects (rachischisis). Absence of bladder control or the presence of deformities of the lower extremities was not considered a contraindication to operation. Age was not regarded as an important factor as to the time of operation: In this series, forty-three of the fifty patients with spina bifida were less than 1 year of age at the time of operation, as were four of the five patients with cranium bifidum. The high operative mortality during this early age period should not be attributed to the operation but rather to the pathologic condition itself, which carries with it an extremely high death rate. In this series three patients were operated on during the first week of life, the nutritional status being considered the determining factor.

Of thirty-six patients who were not operated on and died in the hospital, twenty-three presented hydrocephalus and paralysis of the lower extremities. Autopsy was performed in nineteen cases; hydrocephalus was found in seventeen, in nine of which it was associated with meningitis. In the fifty-seven cases, in which operation was performed simple meningocele was found in twenty; myelomeningocele in twenty-seven; syringomyelomeningocele in three; cranium bifidum with meningocele in four and myelomeningocele in one; spina bifida occulta was found in the two adult patients. In the great majority of the cases of spina bifida (forty-two) the defect occurred in the lumbosacral region.

Operation consisted in the dissection of the sac, with preservation of all nerve tissue and closure of the defect without retention of excess sac tissue but with provision for ample cutaneous and subcutaneous covering. The postoperative development of hydrocephalus was uncommon. Because of standardization and improvement in operative technic, the operative results are arbitrarily considered with reference to those obtained in 1927. Before this date ten of twenty-two patients died; after this date only six of thirty-three patients died. Follow-up study in fourteen cases (eleven of spina bifida with meningocele and three of cranium bifidum with meningocele) indicated that children with these conditions develop normally after operation and rarely have any permanent disability. When nerve tissue is present in the sac in patients with myelomeningocele or syringomyelomeningocele, hydrocephalus and weakness of the lower extremities are frequent complications before operation. Though these symptoms persist it is rather striking that the general idea that hydrocephalus will develop, even if it is not present before operation, does not seem to be supported by the experience of the authors. Almost 50 per cent of their patients developed normally. SPERLING, Philadelphia.

Special Senses

LECTURES ON MOTOR ANOMALIES OF THE EYES. ALFRED BIELSCHOWSKY, Arch. Ophth. **12**: 805 (Dec.) 1934.

This is the first of a series of lectures on motor anomalies of the eyes, and as such is to be considered a physiologic introduction to the reports on motor anomalies as they will appear in subsequent articles. Hering, considered by Bielschowsky to be the master of the physiology of space sense and movements of the eyes, taught that the two eyes should be considered as the halves of a single organ. This applies not only to the visual but also to the motor apparatus. Bielschowsky states that unless the meaning of this doctrine is thoroughly understood it is impossible to avoid mistakes in examining and analyzing the complicated anomalies which are considered in this study and to find the best or, in some instances, the only method for their treatment.

The fundamental law of the visual apparatus concerns retinal correspondence: Each pair of corresponding retinal points has one direction in which the images situated on both points are localized. The principal visual direction (Hering's *Hauptsehrichtung*) belongs to both foveae. Between this principal direction and the visual direction of any other pair of corresponding points is an angle determined by the arc between those points and the foveae. The center of the visual directions is situated either in the region of the imaginary "cyclopean" eye or, if one eye is dominant, in its nodal point. Equal stimulation of a corresponding pair of retinal areas caused chiefly by one particular object outside produces a single sensation. But there are only few images which in near vision can be formed in corresponding parts of the retinas. The images of most objects are formed in noncorresponding (disparate) parts and therefore ought to be seen as double. But actually, as a rule, they are not: If they are situated in the periphery of the visual field one pays no attention to them; if they are very near to the point of fixation they are localized before or behind the plane in which appear the images formed on corresponding retinal points.

The motor apparatus is intimately connected with the visual apparatus. The primary function of the movements of the eyes is to bring the retinal images of objects, which attract attention, to lie in the areas of greatest visual acuity, the maculae. Although these movements are made almost automatically, they are not true reflexes. While true reflexes occur at their best in a state of inattention, ocular movements cease if the attention is diverted; that is, the visual stimuli are conveyed to the cortex, where they induce the ocular movements, while the pathways for true reflexes are subcortical. This statement holds good for every kind of ocular movement induced by sensorial, especially visual, sensations, whether they are in the same direction or in opposite directions (convergent, divergent, etc.).

Hering stated that every motor innervation arising from the cortical centers flows equally to both eyes, causing the associated muscles, either agonists or antagonists, to contract and relax, respectively. It is absolutely impossible to send a motor impulse to one eye only or a stronger innervation to one eye than to the other. Bielschowsky has seen and thoroughly investigated various cases in which this law seems not to apply, as for instance when a single eye can be moved separately up or down. He thinks that these instances do not prove that the movement is produced by unilateral innervation. His explanation is that it is simply some type of heterophoria in which the patient, if he possesses strong fusion power, can overcome the anomalous position of rest of his eyes and maintain binocular fixation for a long time.

One of the most important studies to which the aforementioned law lends itself is that of testing the duction power of the eye. Bielschowsky investigated this fusion power in a long series of experiments, especially the involuntary fusion movements. He states that in speaking of these fusion movements of the eye, vertical divergence both positive and negative as well as divergence and convergence have been erroneously regarded as reflex movements, in that they are independent of the will. He thinks that the only true reflex movements of the eye known are those due to vestibular stimuli arising chiefly from varying postures of the head. He also calls attention to Sherrington's law of reciprocal innervation, which, he believes, is one of the most important laws in ophthalmology, because of its influence on operative procedures

In his discussion of the anomalies of the position of rest, Bielschowsky stresses the importance of anatomic or absolute rest and of functional or relative rest. It is impossible to ascertain an absolute position of rest, because one cannot eliminate the totality of nervous influences. Therefore, one has to be satisfied by ascertaining the relative position of rest as it applies to the position of one eye relative to the other.

Bielschowsky discusses, in part, double or alternating hyperphoria. When one eye is in the primary position, the other is deviated upward. He does not agree with previous observations by other authors that this anomaly is based on an imbalance between the elevator and the depressor muscles. He thinks that the

condition is due to alternating an intermittent nervous excitation of both centers for vertical divergence, the one-sidedness of the vertical movements being based on the coincidence of the voluntary fixation impulse with the involuntary action of one of the vertical divergence centers, so that the fixing eye keeps its position unaltered, as on this eye the innervations of the elevator and depressor muscles neutralize each other. In view of the probable nervous origin of double hyperphoria, Bielschowsky believes that one should not operate on patients with this condition.

SPAETH, Philadelphia.

THE MECHANISM OF VISION: VII. THE PROJECTION OF THE RETINA UPON THE PRIMARY OPTIC CENTERS OF THE RAT. K. S. LASHLEY, J. Comp. Neurol. **59**:341 (June) 1934.

Injuries to the temporal quadrants of the rat's eye were produced by direct incision through the back of the eye. The Marchi method of staining was used to study the terminations of axons in the lateral geniculate body and optic thalamus. The optic tract in the rat is chiefly crossed, but it also contains a considerable number of uncrossed fibers. Before reaching the primary visual center it gives off two small bundles of fibers to the hypothalamus—the anterior and the posterior accessory optic tract. Many fibers of the optic tract end in the lateral geniculate nucleus; some pass through into the adjacent nuclei of the thalamus, but most of them pass over it to end in the thalamus and colliculus. From the extreme temporal margin of the retina, fibers went only to the homolateral lateral geniculate nucleus. From the remainder of the retina all the fibers probably decussated. There was no apparent regularity of arrangement of fibers in the optic nerve or chiasm. The uncrossed fibers occurred chiefly in the inner upper half of the tract. The distribution of the Marchi granules in the ventral part of the lateral geniculate nucleus suggested that they represent only fibers passing through that portion to reach the dorsal portion. In the dorsal portion there was a separate representation of the retinal quadrants, with the inferior quadrants projected above and the temporal quadrants to the inner part of the nucleus. Crossed and uncrossed fibers were traced onto the pretectile and large cell nuclei of the thalamus and seemed to terminate in both. In the contralateral superior colliculus the separate projection of the retinal quadrants was maintained. FRASER, Philadelphia.

THE MECHANISM OF VISION: VIII. THE PROJECTION OF THE RETINA UPON THE CEREBRAL CORTEX OF THE RAT. K. S. LASHLEY, J. Camp. Neurol. **60**:57 (Aug.) 1934.

In this study the occipital region of the cerebral cortex of rats was destroyed, and after intervals of from three to twenty-four weeks the brains were fixed and stained for Nissl substance. Over one hundred specimens were available. These showed retrograde degeneration of the pars dorsalis of the lateral geniculate nucleus. No degeneration was evident except after lesions within the area striata. A previous study has been made on the projection of the retinal quadrants on the primary optic centers. The combined studies show a picture of the projection of different parts of the retina on the visual cortex and show a "cortical retina." There is an approach to point-to-point representation. The most anteromesial part of the striate area represents the dorsal meridian of the eye. Along the lateral margin of the area successive sectors of the temporal quadrants are represented, and the projection of the nasal quadrants continues along the mesial border. Concentric zones of the retina seem to be represented by concentric zones in the area striata. The binocular field is projected to the anterolateral margin of the area. Thus in the rat there seems to be a true cortical retina in which there is not only a separate representation of small retinal areas but a maintenance of the general topographic relations of these areas.

FRASER, Philadelphia.

THE DIFFERENTIATION OF THE PERIPHERAL AND CENTRAL ACOUSTIC APPARATUS IN THE FROG. O. LARSELL, J. Comp. Neurol. **60**:473 (Dec.) 1934.

This study was undertaken in the hope that a detailed survey of the development of the acousticolateral area and its connections in frog larvae and in adult frogs would contribute toward the solution of the problem of the formation of new fiber tracts and particularly would throw light on the differentiation of the lateral lemniscus from the great mass of fibers which arise in the acousticolateral area. The material included 195 series of larval and newly metamorphosed heads or brains of three species of frog stained by a variety of methods and of 60 series of brains of adult frogs sectioned in various planes.

The vestibular root (ventral root of the eighth nerve) and the lateral line roots of the seventh and tenth nerves appear in the larval frog before there is any indication of the dorsal root of the eighth nerve. The lack of dorsal fibers in the eighth nerve appears to be correlated with the underdeveloped condition of the acoustic parts of the labyrinth. At about 9 mm. the first indication of differentiation of the acoustic apparatus appears. At 12 mm. a few dorsal nerve fibers of the eighth nerve are present. After these dorsal root fibers have entered the medulla oblongata the cells of the dorsal part of this region of the substantia grisea send dendritic processes toward the dorsal roots of the eighth nerve and into the lateral line neuropil. As the lateral line system disappears, during metamorphosis, the dorsalmost cells become cut off from connections other than those with dorsal roots of the eighth nerve and become specialized into the dorsal roots of this nerve. The peripheral lateral line nerves disappear by degeneration of fibers and ganglion cells. The superior olive is a new nucleus, phylogenetically, which has been differentiated in connection with the development of a refined type of sensory impulse in the differentiation of the acoustic apparatus from the more primitive vestibular labyrinth. The lateral bulbotectal tract, or lateral lemniscus, is seen arising from cells within the superior olivary nucleus and continues rostrad to end in the corpus posticum (inferior colliculus) and the nucleus isthmi of the midbrain. Additional fibers in this tract seem to come from the dorsal nucleus of the eighth nerve, both homolateral and contralateral, and from the nucleus magnocellularis by way of the arcuate fibers. Cells along the course of these fibers simulate the nucleus of the lateral lemniscus.

FRASER, Philadelphia.

AMAUROSIS ASSOCIATED WITH HYSTERICAL AMBLYOPIA. P. DUPUY-DUTEMPS, Ann. d'ocul. **171**:863 (Oct.) 1934.

P. Dupuy-Dutemps reports a case of amaurosis due to hysteria in a patient with one eye. There was a definite history of simulated nervous disturbances, which were suddenly cured with electrical treatment. Ophthalmoscopic examination gave negative results; the winking reflex and reaction to light were normal. Permanent contraction of the orbicularis muscle and anesthesia of the cornea and face were present on the same side. A case of bilateral blindness of the same nature, which was completely cured, is also reported.

BERENS, New York.

CONGENITAL PARALYSIS OF ASSOCIATED LATERAL MOVEMENTS OF THE EYES WITH UNILATERAL FACIAL PARALYSIS. L. DUPUY-DUTEMPS, Ann. d'ocul. **171**:865 (Oct.) 1934.

L. Dupuy-Dutemps discusses a case of congenital paralysis of associated lateral movements of the eyes with unilateral facial paralysis in which convergence and associated vertical movements of the eyes were normal. The cause was probably agenesis of the nuclei of the sixth nerve and of the right facial nerve. It is unnecessary to explain these paralyses on the hypothesis of supranuclear centers. The oculomotor nuclei, with their connections, assure the coordination of the movements of the eyes.

BERENS, New York.

MONOCULAR NYSTAGMUS AND HOMOLATERAL SPASMUS NUTANS IN AN INFANT. H. ROGER, JEAN SEDAN and N. CARREGA, Rev. d'oto-neuro-opht. **12:** 576 (Sept.-Oct.) 1934.

The cause of this disease has been attributed to (1) monocular amblyopia, especially when there is an associated strabismus, (2) a lesion of the nerve centers and (3) association of monocular nystagmus with spasmus nutans. The patient was a well nourished child, aged 10 months, who at the age of 4 months had osteomyelitis of the right mandible, followed at the age of 6 months by otitis in the right ear. Oscillations of the head in the horizontal plane to the right soon appeared, accompanied by transverse nystagmus to the right, localized in the right eye. The nystagmus appeared especially when the child fixed the gaze on an object. Examination of the eyes showed no abnormality. A caloric test of the left ear produced no nystagmus and did not modify the spontaneous nystagmus in the right eye.

Usually there is a tendency toward recovery. The movements of the head are either in the vertical or the horizontal plane and are increased by fixation of the gaze. Some writers have noted a certain state of spasmophilia; in addition, an important factor is a local cause of irritation. Involvement of the labyrinth may be a factor. Like spasmus nutans, monocular nystagmus occurs in young infants. If in certain cases monocular nystagmus is allied to a mesocephalic lesion, it may be assumed that spasmus nutans is due to the same cause, probably from a neurotropic virus or from an irritative process. The most probable hypothesis is that of an irritative process of the intracerebral labyrinthine pathways that preside over movements of the head and eyes. The monocular character of the nystagmus may be explained by a discrete peripheral paralysis of the nonnystagmic eye or by transitory incoordination.

DENNIS, San Diego, Calif.

CONGENITAL PARESIS OF THE MOVEMENTS OF LATERALITY OF THE EYES TO THE LEFT. A. COLRAT, Rev. d'oto-neuro-opht. **12:**672 (Nov.) 1934.

The case studied was that of a boy, aged 9 years, with paresis of lateral movement of the eyes to the left, which was characterized by nystagmic movements when the patient looked toward the left and by the impossibility of maintaining fixation of the eyes to the left. The automatic reflex movements were abolished or diminished. Congenital ocular paralyses occur in many forms, but no case analogous to the one reported has been found in the literature. As to the nature and seat of the lesion causing congenital paralysis of ocular movements, it is suggested that the anatomic lesion lies at the level of the nuclei of origin of the sixth pair or their fibers of projection.

DENNIS, San Diego, Calif.

Diagnostic Methods

UNILATERAL AMYOTROPHY: ITS DIAGNOSTIC IMPORTANCE FOR CEREBRAL LOCALIZATION. N. W. WINKELMAN and A. SILVERSTEIN, Am. J. Syph. & Neurol. **19:** 58 (Jan.) 1935.

Unilateral muscular atrophy may occur in cases of lesions of the parietal lobe, and in many instances this sign has proved of great diagnostic value. Small and early lesions have been recognized and localized by the presence of unilateral amyotrophy when diagnosis would have been otherwise impossible. The sign seems to depend on the existence of a trophic center in the parietal lobe, for when atrophy develops after the symptoms of motor disturbances have appeared the lesion is likely to be found growing posteriorly, from the precentral gyrus backward. In instances in which the atrophy is the earliest sign and symptoms of motor disturbances are subsequently presented, a lesion located primarily in the parietal lobe, growing anteriorly into the motor cortex, is likely to occur. Mental symptoms indistinguishable from those commonly associated with lesions of the frontal lobe may also occur in cases of lesions limited to the parietal lobe.

Amyotrophy of the parietal lobe is recognized more readily by light palpation than by actual measurement. The muscle first appears to be boggy or soft, and the bony landmarks are frequently more palpable on the amyotrophic side than on the healthy side. Other evidences of involvement of the parietal lobe include incoordination, hypotonia, hyporeflexia, vasomotor changes in the amyotrophic limb and mild astereognosis, graphanesthesia or obtunded muscle-position sense.

DAVIDSON, Newark, N. J.

DEFORMATION OF THE SELLA TURCICA BY TUMORS IN THE PITUITARY FOSSA. K. KORNBLUM and L. H. OSMOND, Ann. Surg. **101**:201 (Jan.) 1935.

The roentgenologic manifestations in seventy-four verified cases of intrasellar tumors were critically analyzed. Definite deformation of the sella turcica occurred in every instance (100 per cent). The changes produced in the sella turcica by intrasellar tumor depend on the nature of the tumor, the rapidity of its growth and the direction of its extension. In approximately 75 per cent of the cases in this series and in 82 per cent of the cases of adenoma, the deformation of the sella turcica was sufficiently characteristic to be regarded as pathognomonic of a tumor of the pituitary gland.

GRANT, Philadelphia.

THE PHYSICAL DYNAMICS OF ENCEPHALOGRAPHY. PURCELL G. SCHUBE, J. Nerv. & Ment. Dis. **80**:291 (Sept.) 1934.

Though much has been written about the technic of, and the indications and contraindications for, encephalography, nothing has been said concerning the physical dynamics involved. Schube studied the various physical factors involved in encephalography, assuming that in the carrying out of the process the fluid was replaced by an equal quantity of atmospheric air, the temperature of the body was 98.6 F. and that of the air was from 65 to 80 F. Atmospheric pressure should vary from that at sea level to that at an elevation of 1 mile. Reasoning from the kinetic theory of gases Schube declares that regardless of the amount of spinal fluid removed or of the amount of air injected the increase in the intracranial pressure is always the same as long as the internal temperature of the patient remains constant. Any deviation from these conditions in any respect alters the resulting pressure in a direction dependent on the altered factor.

In many cases the spinal fluid pressure is either greater or less than that of the atmosphere. In either case, after the encephalographic procedure the pressure of the air in the cerebrospinal fluid space is greater than that originally exerted by the cerebrospinal fluid because of the rise in temperature from 65 F. to the body temperature of 98.6 F. This pressure progressively diminishes as the temperature of the injected external air approaches that of the body. It is probable then that the period of increased intracranial pressure, severe headaches, vomiting, sweating, cardiac irregularities and convulsions may result, but they can be avoided by two methods: (1) preheating the air to body temperature before injection and (2) injecting only sufficient air of room temperature to produce a final intracranial pressure equal to the atmospheric pressure.

HART, Greenwich, Conn.

CALCIUM IN EPILEPSY. G. M. GRIFFITHS, J. Neurol. & Psychopath. **15**:29 (July) 1934.

From a study of blood calcium in forty-eight patients, mostly males, in an epileptic colony the following conclusions are drawn: The average serum calcium level falls within normal limits, though the range of values for the group and individual patients alike, is wide and variable. The average of values is greater before a seizure than after; the highest figures are obtained during, and low values exist after, a series of attacks. There is an inverse relationship between the serum calcium value in the case of seizures in the day and those in the night. The level for serum phosphorus was also found to be within normal limits, though

it exhibited a wide range of variation. The calcium-phosphorus ratio was not always disturbed by attacks, though abnormal ratios occurred in association with them. Diminution of serum calcium by sesquiphosphates is detrimental; treatment with large doses of calcium is not necessarily, but may be, beneficial.

SPERLING, Philadelphia.

THE SERODIAGNOSIS OF SYPHILIS. R. W. FAIRBROTHER, Lancet **2**:701 (Sept. 29) 1934.

The use of two approved methods of examination as recommended by the International Conference on the Serodiagnosis of Syphilis at Copenhagen (1928) and Montevideo (1930) is supported by tests performed on 1,500 serums, of which 700 were from nonsyphilitic patients. The routine Kahn test and a modified Wassermann test were used by the authors, the main variation of the latter being the use of dilutions of serum of 1:2, 1:8 and 1:32. Such a double examination appears to be of paramount importance in dealing with serums giving doubtful or unsatisfactory reactions by any one method. It was found that if either the Wassermann or the Kahn test alone had been employed a small number of false negative reactions would have been recorded. The number of serums from persons with definite syphilis giving a doubtful or positive reaction to the Kahn test but a negative reaction to the Wassermann test was 19, while the number giving a doubtful or positive Wassermann reaction but a negative Kahn reaction was 51. These figures indicate that the modified Wassermann test, which is definitely more sensitive than the method formerly employed, is also more sensitive than the Kahn test; this applies particularly to patients undergoing intensive treatment. Whether this fact counterbalances the relative simplicity of the Kahn test as compared with the complicated Wassermann test is an open question. The reading of the Kahn test is usually simple, but in a few instances the differentiation between a negative and a doubtful reading presents some difficulty.

When only one routine laboratory test is carried out for the serodiagnosis of syphilis, the modified Wassermann test should be employed. The Kahn test should, however, always be available for serums giving irregular readings.

BECK, Buffalo.

Society Transactions

NEW YORK NEUROLOGICAL SOCIETY

Regular Meeting, April 2, 1935

ISRAEL S. WECHSLER, M.D., *President, in the Chair*

ABNORMAL ASSOCIATED MOVEMENTS OF THE EYELID (PSEUDO-GRAEFE SIGN). DR. MORRIS B. BENDER.

Downward rotation of the eyeball is normally associated with a downward movement of the eyelid. When an attempt is made to look downward with the eyes closed, the eyelids open slightly and assume the position as in downward gaze. An unusual abnormal associated movement of the eyelid is illustrated by the following case:

A telephone operator, aged 20, was admitted to the Mount Sinai Hospital in February 1934, with the chief complaints of drooping of the left eyelid, double vision and headache. She had been in good health until nine months before admission, when a severe cold in the head developed, with redness and swelling around the left eye. The latter signs persisted for a few days and were soon followed by drooping of and difficulty in opening the left eyelid. At the same time mild headaches occurred in the left supra-orbital and temporal regions. These recurred daily and lasted from one to three hours, during which time the eyes "felt heavy." A few weeks after the onset of the illness the patient noted impairment of vision in the left eye. In spite of the persistence of these symptoms, she continued to work. Three weeks before admission she began to see double on looking to the left. At the same time she became aware of nausea and dizziness with sharp changes in position of the body. She finally had to give up work because of the progressive nature of the condition and was admitted to the hospital.

Physical examination showed no evidence of disease of any of the internal organs; there were some thinning of the scalp and tenderness to percussion in the left frontotemporal region. The neurologic status was normal except for: (*a*) incomplete ptosis of the left eyelid; (*b*) incomplete paralysis of all extra-ocular muscles on the left side; (*c*) a very sluggish response of the left pupil to light and in accommodation and impairment of the right to left consensual response, and (*d*) visual acuity of 20/30 in the left eye and 20/20 in the right eye. The peripheral, central and color fields were normal in both eyes. Laboratory tests gave normal results. Lumbar puncture showed an initial pressure of 120 mm. of water; the total protein content of the spinal fluid was 51 mg. per hundred cubic centimeters; Wassermann tests of the blood and spinal fluid were negative. A roentgenogram of the skull showed a calcific mass in the middle fossa to the left of the sella turcica in the region of the posterior clinoid processes, suggestive of calcification in a tumor. The sellar outline was not clear. There was some evidence of increased intracranial pressure.

Eight days after admission a craniotomy in the left frontotemporal region was performed, and a calcified tumor extending laterally from the sphenoid ridges to the sella was partially removed. The diagnosis based on the pathologic picture was osteochondrosarcoma. The postoperative course was uneventful. Three weeks after craniotomy slight improvement in the muscles of the left eye was noticed.

Three months later the following signs were present: (*a*) paralysis of the superior and inferior rectus and oblique muscles on the left; (*b*) middilatation of the pupil and fixation to direct and consensual stimulation and in accommodation; (*c*) absence of ptosis, although the right palpebral fissure was slightly greater than the left, and (*d*) ability to close the eyelids at will and with equal force on both sides. When the patient looked down, however, the left eyelid remained

behind while the left eyeball turned slightly inward. When she attempted to look down when both eyelids were closed, the left eyelid snapped upward and assumed the same position as in looking forward. The right eyelid showed the normal associated position and movement. This phenomenon, known as the pseudo-Graefe sign, is not common. Often the history in such a case is that of a tumor, infection or trauma involving the peripheral portion of the third cranial nerve, first manifesting paralytic symptoms and later giving rise to the phenomenon described.

A tentative explanation for the pesudo-Graefe sign is that in regeneration of a damaged nerve trunk the axons reach the peripheral stump in great disorder, entering the nerve sheaths of different nerve bundles. Under such conditions an impulse originally directed to one muscle will pass to another, producing a contraction in the muscle or group of muscles for which it was not designed. Thus, in this patient the partially regenerated fibers of the third cranial nerve trunk innervate chiefly the internal rectus muscle and the levator palpebrarum. The axons which were supposed to have entered the nerve sheaths of the nerve bundle supplying the inferior rectus muscle regenerated in the wrong direction and spread into the nerve sheaths of bundles supplying the levator and internal rectus muscles. An impulse meant for the inferior rectus muscle now reaches the levator and internal rectus muscles, producing a slight contraction in these muscles. At the same time, the relaxation of the levator muscle, which is supposed to be present when the eyelid moves downward, does not take place. It is also probable that fibers from other bundles of the third cranial nerve entered the "regenerated pathways," producing slight internal motion of the rectus muscle on upward movement.

This is similar to the explanation of facial contracture and spasm following Bell's palsy.

BENIGN TUMOR OF THE SPINAL CORD SIMULATING A MALIGNANT GROWTH: REPORT OF TWO CASES. DR. IRA COHEN.

CASE 1.—A man, aged 68, on roentgen examination presented evidence of a prepyloric defect: The contents of the stomach during fasting showed blood and an absence of free hydrochloric acid. The patient entered the hospital because of pain and marked weakness in the legs and sphincteric disturbances. Of four spinal punctures, the first and fourth showed block, with a high protein content of the spinal fluid, and the second and third showed no block, with a lower protein content of the spinal fluid. The sensory level was indefinite. The level was determined by means of iodized oil at the second thoracic vertebra. Spinal metastasis from a gastric carcinoma was first considered. An anteriorly placed hemangioendothelioma was removed by laminectomy.

CASE 2.—A woman, aged 67, had weakness and pains in both legs, which had been progressive for two years. She was admitted to the hospital with complete paraplegia and signs of a lesion at the level of the eleventh thoracic segment. A roentgenogram of the spine showed fusion of the eighth and ninth thoracic vertebrae, and when gastric analysis revealed complete achlorhydria and blood a malignant condition of the stomach with secondary spinal metastasis was considered. However, a roentgenogram following a meal of barium sulfate showed a para-esophageal hernia of the stomach through the diaphragm. At laminectomy an anteriorly placed meningioma was removed from opposite the ninth thoracic vertebrae.

DISCUSSION

DR. BERNARD SACHS: This seems to me to be rather a coincidence—two cases of benign tumors in which a malignant condition of some other organ was suspected. The point which is important to bear in mind is that an unusually large number of tumors of the spinal cord are benign. One should be more optimistic regarding the nature of this condition.

DR. LEO DAVIDOFF: In reference to the first case, the histologic nature of the tumor is important. The fact that it is a hemangioblastoma and that the patient

has some type of lesion of the stomach is suggestive of multiple hemangioblastoma or Lindau's disease, and I think that later developments may show that the patient has other tumors elsewhere. At least this possibility should be kept in mind.

CONVERSION HYSTERIA WITH AMNESIC FEATURES. DR. S. E. KATZ.

V. M., an American-born housewife, aged 47, was admitted to the Psychiatric Institute on Nov. 5, 1932; except for three short periods of parole at home, she has remained in the hospital up to the present time. In May 1932 radium was implanted for metrorrhagia; an artificial menopause resulted. Following this there developed paranoid ideas that people in the church were "framing" the patient, that she was being accused of having stolen a pocketbook from a parishioner and that she was being constantly watched; she refused to leave her apartment. Soon the paranoid ideas involved her husband. He was in league with other people and was trying to get rid of her. Then there developed delusions that the bed in which she was sleeping was charged with electricity and that the food was being poisoned. She complained of headaches, pain in the spine and over the pelvic region and numerous other somatic disturbances, because of which she was admitted to the New York Hospital, where she remained for two weeks. No organic basis for the symptoms was disclosed.

On admission the patient presented a picture of retarded depression and offered little information about herself. After a few days she preferred to remain in bed; when an attempt was made to get her up, she would fall to the floor. She became mute.

Because of poor cooperation physical examination was incomplete, but there was no evidence of any muscular weakness. Two weeks after admission the patient indicated by gesture that she was having pain over the entire right side of the body. It was found then that she had paresis and complete hemianesthesia on the right side. The patient indicated that she was unable to use her right arm, although she did use it quite well when she was alone or with other patients. The hemianesthesia is still present and includes one half of the tongue. She still has paresis of the right arm but walks well.

For the next three months she remained practically mute. Then gradually she began to communicate by writing and later verbally. She expressed ideas of unreality—that the world did not look real to her; she could not recall entering the institute. Gradually the amnesia extended over a longer period, finally including her entire life. In March the patient stated that she did not remember anything about herself up to a few days before when she was standing at a window and saw people on the drive. She began to wonder where she was—"It seems as if I dropped into a world. Where or what I was before I don't know. I didn't know what water or trees were or the patients or nurses." She stated that the nurse had to explain to her that "this was a hospital." At first she thought that she was in a school and that the doctors were teachers and the nurses matrons. She did not remember her father or mother or whether they were living or dead, nor could she remember ever having been married or having raised a child. The person who was introduced to her as her husband appeared strange to her, but his voice was familiar; she would not have known that she had a son if she were not told so by the nurses. She stated that all the activities that she is able to carry on now on the ward she learned from the nurses or other patients, that she learns very quickly and in that way has picked up writing and fancy needle work. The patient stated that she feels much younger than the age she was told she is and that she feels more at home with girls of 15 or 16, although she is embarrassed by the marked physical difference between herself and them. She would like to remain here indefinitely because everybody is so kind to her. The patients feel that under the tutelage at the institute she could "grow up" very quickly. She has learned a lot about sex from other patients (girls of 15 or 16).

Little is known of the patient's early childhood. She left school at the age of 13, having reached the seventh grade. She worked as a salesgirl in a 5 and 10 cent store, and then became a "little cadet" in the Salvation Army. She was quite religious. Her health was good. Menses were established at 14; there was some dysmenorrhea. For the past fifteen years she suffered with marked headache during the catamenia. At the age of 24 she married a man whom she met in the Salvation Army. Prudish and frigid, the sexual act was extremely repugnant to her. One pregnancy resulted. She had several operations for gynecological conditions; artificial menopause followed treatment with radium.

At the onset of menstruation, about the age of 14, the patient was frightened by a Negro. She lost consciousness and was sent to the Presbyterian Hospital, where, it is said, she remained in a state of stupor for three days. This episode could not be verified.

She was described as stubborn, religious, suspicious, excessively modest and fairly sociable, having few close friends.

The patient's father is living at the age of 81; he uses alcohol excessively and has a violent temper. The mother died of peritonitis when the patient was in her teens. She is the second of four siblings. One brother is addicted to the use of alcohol. Another brother, also a heavy drinker, accused his wife of infidelity, killed her and then committed suicide.

DISCUSSION

DR. PAUL SCHILDER: This is an impressive case. One has the feeling that such cases no longer belong to this century. This is the type which was described frequently about 1890; by 1900 they had become rarer.

Perhaps the patient was impressed by a story which she read when she was young. Perhaps in this way her conflicts found their particular form of expression. Why does a person choose a particular type of expression for his or her neurosis? That is the chief problem in this case. Why are these cases becoming so rare? In the Bellevue Hospital one observes many cases of amnesia, which passes off quickly. Lasting a longer or shorter time in different cases, in the great majority it disappears in a few days. The old-fashioned amnesia persisted for a long time. Such cases are described by Ribot in "Diseases of Memory" (New York, D. Appleton & Company, 1882) and "The Diseases of the Personality" (Chicago, Open Court Publishing Company, 1881).

I do not think that there can be any doubt that this is a disturbance of the hysterical type. Hysteria is generally regarded as a neurosis. One may speak of a hysterical psychosis when the term psychosis is used in the sense of social maladaptation. There are no definite paranoid trends. The patient has all the agreeable manners which only the hysterical patients have. She shows also a sufficient amount of transference. The symptoms in the beginning of the illness are interesting from a psychoanalytic point of view; something was said about a stolen pocketbook and also about an artificial menopause. There may be a connection between the two. I am rather convinced that there is; perhaps Dr. Katz has a little to add in this regard. Of course, one cannot actually describe the psychogenesis in a case such as this. Seemingly the patient's family is one in which there are conflicts, which are built up in a more or less dramatic way. The underlying conflicts have probably to do with the menopause. It is difficult to say what the prognosis may be in such a case. The patients are comparatively happy as long as they are hysterical; when they recover, they are probably unhappy.

DR. S. E. KATZ: I purposely limited the presentation to the factual clinical material because anything that might be said regarding the psychogenesis of the symptom complex would be highly speculative. It is true that the patient has been in the hospital for two years, but what has been learned about the mental mechanism is very meager. She has not responded to any form of psychotherapy, meeting all questions with "I don't know" or "I was told so by the nurses" or "I do not understand your question." She resists hypnosis; I was unable to get any additional information from her while she was under the influence of sodium amytal.

The Kent-Rosanoff association test with key words was given her. Here again, she was constantly on her guard and gave bizarre responses to the key words. It is interesting to note that there was no delay in the reaction time. Her responses to other words were 60 per cent psychotic.

The only plausible explanation that I have to offer is that this woman has responded to an unbearable life situation, first by a depression and later by a complete annihilation of her environment and herself. As a result, she is a changed person; the people are not the same, and the whole world looks different to her. As indicated in the brief abstract, the patient is poorly endowed by constitution and heredity, to which very taxing environmental factors, namely, a high-strung, intolerant husband, are added.

DR. ISRAEL STRAUSS: Dr. Katz thinks this is not a neurosis but a psychosis?

DR. S. E. KATZ: I did not say so.

DR. ISRAEL STRAUSS: Dr. Katz used the word psychosis, but perhaps he agrees with Dr. Schilder and thinks it a neurosis.

DR. S. E. KATZ: No, that is another interesting feature of the case. It is not a very clearcut case of neurosis or psychosis. It is a classic picture as far as the conversion hysteria is concerned, and there are also definite paranoid ideas with delusions. These are the facts; how to fit them into a single disease picture I do not know.

DR. JOSHUA ROSETT: Dr. Schilder and Dr. Katz agree on one point. They both think that the patient's trouble is the expression of conflict. They do not give any reason for this. I should like to know why they are so certain of it and of what nature is the particular conflict in this case?

DR. PAUL SCHILDER: Is the Babinski sign an evidence of a lesion of the pyramidal tract?

DR. JOSHUA ROSETT: It is. How does a conflict explain the hysterical conversion symptom?

DR. PAUL SCHILDER: I do not think this case is one of neurosis, if one uses the term in the social sense of a comparatively good adaptation. I think it is one of hysteria. The term neurosis is not a diagnostic term; it is a social term, and many conditions which are called hysteria are considered to be psychoses from a social point of view. The patient is in a psychiatric institute, but still I do not doubt that the diagnosis of her condition is hysteria.

To reply to Dr. Rosett, a conversion symptom is a sign of the presence of a conflict in the same way as the Babinski sign is a sign of a lesion of the pyramidal tract.

DR. JOSHUA ROSETT: The difference is that in patients exhibiting a Babinski sign degeneration of the pyramidal tract is observed post mortem. No one has even noted a conflict post mortem.

SURGICAL TREATMENT OF MÉNIÈRE'S SYNDROME BASED ON NEW RESEARCH IN THE PHYSIOLOGY OF HEARING. DR. JOHN GUTTMAN.

It may be assumed with Wegel (*Arch. Otolaryng.* **14**:158 [Aug.] 1931) that tinnitus (subjective noises), one of the main symptoms of Ménière's syndrome, is present all the time, even in normal ears, and is produced by some metabolic process. In a normal ear one is ordinarily not aware of these noises, because they are masked by outer noises (Guttman, John, and Ham, L. B.: *Arch. Otolaryng.* **12**: 425 [Oct.] 1930). In a deafened ear, however, as in Ménière's syndrome, the outer noises are not heard because of the deafness; thus the masking effect is absent, and the patient becomes aware of the inner subjective noises which are called tinnitus.

From the work of Wever and Bray (*J. Exper. Psychiat.* **13**:373, 1930) it is known that when the ear of a cat is stimulated by sound the resulting nerve impulse in the auditory nerve is accompanied by an electrical action current. I

have shown in previously published experimental work (*Laryngoscope* **43**:983, 1933) that stimulation of the inner ear produces two distinct types of electrical disturbance. One of these is of the action current type and is present in the auditory nerve. The other is of a different type and originates in the cochlea, not in the auditory nerve or in the endings of the auditory nerve. Barrera and I (*Am. J. Physiol.* **109**:704, 1934) have shown that degeneration of the nerve and nerve endings in the cochlea fails to destroy this second cochlear type of electrical potential. This cochlear electrical disturbance is probably the cause of tinnitus. The operation here described has for its aim the removal of this cochlear electrical disturbance.

The operative procedure consists in the main of severing the tendon of the stapedius muscle, incision of the secondary tympanic membrane and draining off the perilymph of one or both scalae. The pitch of the sound caused by tinnitus, whether of high or of low frequency, and the severity of giddiness accompanying the tinnitus will influence the operative procedure in each case. Sixteen patients with Ménière's syndrome have been operated on to date, with partial or total cure in about 50 per cent. These patients had been recalcitrant to all therapeutic measures for years.

DISCUSSION

DR. LEO DAVIDOFF: Dr. Guttman's presentation is extremely interesting. As I understood him, he spoke of a metabolic disturbance of the cochlea which is responsible for this syndrome. This metabolic change has been investigated by Furstenberg and Newbergh in the clinic at Ann Arbor. They discovered that the change is due to an abnormality in the concentration of the sodium ion and treated patients with Ménière's syndrome by limiting the intake of sodium. Their results are impressive. Their figures correspond approximately to those of Dr. Guttman —about 50 per cent of the patients recovered, and a considerable proportion of the remaining patients were remarkably improved. I should like to have Dr. Guttman's opinion on that work.

DR. ISRAEL STRAUSS: I do not know whether I understood Dr. Guttman correctly. I know of the work of Dandy and MacKenzie, who sever the eighth nerve in cases of Ménière's syndrome, particularly in severe cases. Did I understand Dr. Guttman to say that they were not successful in 50 per cent of the cases?

DR. JOHN GUTTMAN: Yes.

DR. ISRAEL STRAUSS: If I understood Dr. Guttman correctly, he means that when he destroys the cochlear apparatus he can cause the tinnitus to disappear. These symptoms first have to reach the brain through awareness. If one cuts the acoustic nerve, why should any of these symptoms continue? That is what I cannot understand.

DR. JOHN GUTTMAN: In reply to Dr. Davidoff, I know of the report of Dr. Furstenberg; it may be that there is something to it.

Dr. Strauss is perfectly right; the matter is not understood. Not enough is known about the entire mechanism of the cochlea to enable one to state exactly the reason that the symptoms persist when the connection of cochlea and brain is severed. I tried to suggest an explanation; there may be some nerve other than the eighth involved in connecting or transmitting these impulses to the brain. It may be a sympathetic nerve or some other branch of the autonomic nerve system. I am unable to prove this, and it is merely a suggestion.

DR. HENRY ALSOP RILEY: Does not the answer to this problem lie in the fact that there are many types of Ménière's disease? There are Ménière's disease of the peripheral apparatus, Ménière's disease of the conductive apparatus and Ménière's disease of the receptive nuclear apparatus. The fact that a certain number of patients are relieved by section of the auditory nerve indicates that the condition in these patients is either in the peripheral part of the conducting apparatus or in the peripheral receptive apparatus. In patients unrelieved by this procedure the condition is in all probability due to irritative processes within

the medulla and the brain stem. It is known that the region of the receptive cochlear and vestibular nuclei is very richly vascularized. There are a considerable number of relatively large veins immediately overlying the vestibular and cochlear nuclei. These may easily be dilated; there may be some change in them that gives rise to abnormal responses of the receptive apparatus. Many other processes, such as gliosis, inflammatory reactions, etc., may give rise to the symptoms of Ménière's syndrome also through central irritation.

DR. ISRAEL STRAUSS: I think that Dr. Riley "hit the nail on the head" when he tried to explain the difference in results, because the question of selecting what is Ménière's syndrome among these different cases is important. I have observed one interesting phenomenon in these cases. If one applies the caloric tests to a patient, vertigo, nystagmus and nausea result. I do not think one causes tinnitus. In cases of so-called Ménière disease there are tinnitus, some difficulty with hearing, nausea and vertigo. I find that the degree of hearing in many cases may be minimal; sometimes it is not affected, but the remarkable fact remains that if one tests these patients calorically one is not able to demonstrate any abnormal responses on the part of the vestibular apparatus.

DR. JOHN GUTTMAN: The vestibular reaction is normal, and so are the results of inspection of the ear.

OPHTHALMOSCOPIC APPEARANCE OF THE OPTIC NERVE HEAD OF THE NEW-BORN INFANT. DR. SAMUEL KARELITZ (by invitation) and DR. PETER VOGEL (by invitation).

On ophthalmoscopic examination the majority of the optic disks of new-born infants show a grayish color of variable degree and hue. This may suggest atrophy of the optic nerve to the inexperienced observer, as it has in several cases known to us. The grayness may persist up to 6 months of age. In general, it is more marked in blond babies and most striking in albinos. Very dark babies show this finding in lesser degree. The Negro infant's optic nerves appear more like those of the adult.

We believe that lack of pigmentation in the fundus and poor development of the vascular tree in and about the nerve head are the most important explanations of these findings. Other explanations are referred to as possible contributing factors. Because of this normal postnatal developmental phenomenon, one must be cautious in diagnosing atrophy of the optic nerve on the finding of gray nerve heads.

Reference is made to other incidental findings in the eye of the new-born infant.

DISCUSSION

DR. ROBERT LAMBERT: There does not seem to be any doubt that Dr. Karelitz and Dr. Vogel have brought out important facts which may be useful in sparing a certain amount of anguish to parents. In making a diagnosis of atrophy of the optic nerve in an adult one has the added advantages of subjective testing of visual fields, blindspots and visual acuity, whereas in a child one has to rely on the possible perception of light and the movements of the eyes following the light. I do not think that one can describe colors accurately to another person, and in this work Dr. Karelitz and Dr. Vogel and I have frequently had slightly different opinions as to whether the disk was gray, bluish gray or slate gray. I think that it behooves any one who is interested to look at infants' fundi and establish in his own mind what the normal variations are. There are no perfect means of colorimetrically measuring the retina; although there have been a few ophthalmoscopes equipped with an apparatus for comparing colors, they are not practical.

I am fully in agreement with Dr. Karelitz and Dr. Vogel that the probable explanation lies in a lack of pigment in the fundus. It is well known that the iris is light blue or violaceous at birth, and within a few months it may become quite brown, the difference in color being due to the amount of pigment laid

down. The same changes take place in the posterior layers of the eye. This is not really a difference in the color of the nerve head but a difference in appearance due to the color around it. Myelinization of the nerve is not complete until about the tenth week, but I cannot see how that would influence the appearance of the nerve head to such an extent.

An interesting feature of the report of Dr. Karelitz and Dr. Vogel is the low incidence of retinal hemorrhages as compared with other series of cases reported. This speaks well for the type of obstetrics which is practiced in this particular institution.

Dr. Bernard Sachs: This is an interesting and worthwhile investigation. I feel much encouraged by the conclusions that the authors have reached. I have had occasion to examine the eyes of many infants in families in which amaurotic family idiocy had occurred in one child, and I have seen other children and examined them in the earliest periods in order to find out as early as possible whether there was a likelihood of the development of this disease. I must confess I have had great misgivings about the proper interpretation to give to these early retinal pictures. I agree with Dr. Lambert that it is difficult indeed to describe these colors accurately. While this paper was being presented I was wondering what was meant by a grayish appearance of the nerve head, and when I saw the pictures I would have called many of them blue rather than gray, but I felt that after all I had seen something very similar. Whenever I saw a blue head of that sort I felt I would have to predict the later development of atrophy of the optic nerve. I am certain that in some of these patients whom I have been able to follow it did not develop; in spite of the early bluish appearance in that particular child the ophthalmoscopic picture of amaurotic family idiocy did not develop. It is comforting to know that these early discolorations may be within normal bounds.

Dr. P. Vogel: I have nothing to add except a practical point. When we first started with this work we found it difficult to examine the eyegrounds. A baby 1 or 2 days old is difficult to manage. We found that if we made this examination immediately after nursing the baby is practically asleep for the examination. That is a practical point which may be helpful in making the examination a great deal easier.

Dr. Samuel Karelitz: I think that some may want to know whether we dilated the eyes. We did, because we found that most of them had pupillary reaction to light. We used a 2 per cent solution of homatropine hydrobromide and observed no ill effect from it.

Dr. Lambert's innovation of lid retractors was excellent and facilitated our work greatly.

Since this report was written I know of two more cases in which the condition has been wrongly diagnosed as atrophy of the optic nerve because of this peculiar discoloration of the nerve heads.

The Dynamics of Group Psychotherapy and Its Application. Dr. Louis Wender.

The paper is based on six years' experience with group psychotherapy at the Hastings Hillside Hospital and covers a review of hospital conditions that prompted the adoption of this method. A definition of this method of therapy, an interpretation of its ideologic basis, a description of its application and scope and an evaluation of results are given.

I define group psychotherapy as the application of some of the hypotheses and methods of psychoanalysis, in combination with intellectualization, which when applied to a group for purposes of treatment under conditions of active therapeutic control, will lead to the release of certain emotional conflicts and a partial reorganization of the personality and ultimately to an increased capacity for social amalgamation.

A classification is presented of the types of patients who respond to this type of therapy. Methods of procedure are analyzed, and the content of sessions is described.

The dynamics operating in group psychotherapy are described as:

1. Intellectualization. It is stressed that intellectual comprehension is a factor in emotional acceptance and that it plays a significant rôle in the leading process.

2. Patient-to-patient transference. It is indicated that this type of transference, with its component of identification, helps to enhance transference toward the therapeutist and that its greater permanence furthers the socialization of the patient.

3. Catharsis in the family. The group set-up with the therapeutist acting as a parent reenacts an early family situation and serves as a means of resolving early traumas in child-parent relationships.

4. Group interaction. Group interaction, a phenomenon which exerts a universal and constant influence, is utilized in formulating a changed perspective toward behavior. This results in a lessening of personal tensions and a more objective evaluation by the patient of his own problem. Under the group motivation new ego strivings are inculcated, the patient's drive to recover gains impetus and the feasibility of recovery becomes convincing through observation and intimate knowledge concerning other patients.

The wider use of this method is advocated in hospitals which receive patients amenable to psychotherapy.

DISCUSSION

DR. A. A. BRILL: I have enjoyed listening to Dr. Wender's paper; some of his views sound plausible and interesting and some more or less theoretical. I can understand, however, why he could not give more clinical material to demonstrate the points that he makes. I have always believed that whatever benefits the patient is worth doing, and I also know that group psychotherapy, so-called, has been practiced by many in this country and abroad for many years. Perhaps the first person who practiced it in this country was Dr. Josephine Jackson, of California. She did not work out her method into a system such as Dr. Wender presented, but she would select reprints or psychologic subjects written by various physicians and read them to a group of her patients, who then had a general discussion. Some of her patients talked to her about various problems of special interest to themselves. She claimed good results from such psychotherapeutic procedure. I think that Dr. Wender's method could be beneficially applied only to the type of patients he mentions, namely, those with mild psychosis and psychoneurosis. I have seen, however, a few patients treated in groups who have not benefited by this treatment. I have in mind two patients who, instead of being helped by identifying themselves with other patients, took on additional symptoms. Dr. Wender is fully aware of such possibilities and knows how to avoid them.

As far as the actual technic is concerned, Dr. Wender's procedure seems to me the first systematic and logical presentation of this kind of therapy. Dr. Wender knows what he is doing, while the others are either too loose and haphazard or are lost in theories. Dr. Wender's technic is logical because his psychotherapeutic principles are taken mainly from psychoanalysis. From what he said, he also uses suggestion and persuasion. Although I am a firm believer in psychoanalysis, I believe that it is important that something should be done for patients in sanatoriums and hospitals, where, as Dr. Wender pointed out, the patients cannot be treated on a pure psychoanalytic basis. There are a great many practical reasons why, in my opinion, psychoanalysis, as such, cannot be carried out in small sanatoriums. Dr. Glueck, who has had considerable experience with this problem, will, I hope, speak about it. Dr. Wender makes an effort to socialize the patients because these patients had to come to the hospital because they were in conflict with the group; they needed reintegration. In addition, he also gives them some information about the mental processes which are the bases of their conflicts, concerning which they knew nothing.

DR. BERNARD GLUECK: I think that Dr. Wender is an outstanding therapeutist, and his attempt to formulate his approach in a somewhat disciplined manner is a valuable undertaking. All who are confronted with the task of doing something for the hospitalized patient realize how difficult it is, even under the best conditions, to do complete justice to the situation by way of adequate individual therapy, and any approach that mobilizes therapeutic equipment in such a way that it could be applicable in group fashion deserves serious consideration. There are, however, certain features about the situation that Dr. Wender has to deal with which are radically at variance with what one encounters in the ordinary hospital for persons with mental disease. I think that these differences should be pointed out in case some one might be encouraged to try the same procedure and fail to obtain the results claimed for it by Dr. Wender.

In the first place, Dr. Wender is fortunately placed in being able to select his patients rigidly. The institution offers a high type of treatment for a negligible charge, as well as entirely free care; it has a great many more applications for admission than it can accommodate, which gives an opportunity for the selection of patients who promise the most from a therapeutic point of view. In the second place, this is a Jewish hospital serving a certain fairly well defined social stratum. The more or less uniform social-economic background of these patients, as well as the common traditions of a unitary racial group, promote and facilitate the adaptabilities of the patients to group functioning. An important element in cases of psychoneurosis, as well as in many cases of early psychosis, is the painful feeling of isolation. The opportunity that these patients find at Hastings for a ready assimilation into the group must do a great deal toward eliminating this feeling of isolation and substituting for it a sense of belonging. This certainly must have a great therapeutic value and help toward personality reintegration.

There is much practical wisdom in what Dr. Wender has said, and I think that he exhibits a great deal of courage in his attempt to formulate his group therapy procedure. It is a type of courage that can come only from a direct, honest and, one might say, charmingly ingenuous approach to the problem of psychotherapy; I hope that Dr. Wender will be able to extend the usefulness of his method to increasing numbers of patients.

I would rather not enter into a detailed discussion at this time of the theoretical implications of the method, except to state that Dr. Wender would be more accurate in referring to preconscious rather than to unconscious material, because of the readiness with which this material is brought to the surface in the group sessions. I do not believe, however, that this is of great significance as regards the problem as a whole. At any rate, those who have been in close touch with the Hastings Hillside Hospital know that it has an enviably high record of positive therapeutic results, and while one cannot minimize those special factors which characterize the situation at this hospital, it is not difficult to appreciate the significant rôle which Dr. Wender himself plays in achieving this excellence.

DR. THEODORE DILLER, Philadelphia: Benjamin Rush published his remarkable book on insanity in 1812; he died the next year. In this book he set forth a plan for group psychotherapy of which that outlined by Dr. Wender is but an elaboration.

All the members will recall that Rush was a signer of the Declaration of Independence. He was a man of many parts. He was very humane, opposed to slavery and also to the use of alcoholic drinks. He was a great friend of prisoners and advocated prison reform and the abolition of the death penalty, which at that time was enforced for five or six different crimes. He had given a great deal of thought to the subject of education and was chiefly responsible for the foundation of Dickenson College.

Rush joined the faculty of the University of Pennsylvania in 1769 and remained on it until his death, in 1813; he probably taught 3,000 students. For many years Rush took a great interest in the subject of mental disease, and while his book contains many fantastic assumptions and theories and observations that are vague and foolish, it also contains much of real value. For instance, Rush has presented

in appealing attractive form group psychotherapy. He organized a scheme at the Pennsylvania Hospital whereby some person was appointed to act as a friend and guide to a group of patients with mental illness. This person was to concern himself in their welfare, read, play games, talk and engage in occupation with them. For the group he was friend, adviser and general mixer.

DR. ISRAEL STRAUSS: I am in hearty agreement with everything that has been said in the discussion of Dr. Wender's paper. It might be well for some who are not familiar with this hospital to be informed that this institution has a capacity of 40 patients, with 3 psychiatrists in attendance. The patients are highly selected. No patient with an incurable mental illness is received; in fact, no person who should be committed to an institution. As Dr. Glueck has pointed out, the patients are principally Jewish, although there have been others at the institution, since it is nonsectarian. They find the hospital as they have called it: "The Hospital with a Heart."

Dr. Wender in describing this method claims no originality for it. He is calling attention to the usefulness of the method in selected cases. The turnover of this hospital, and by that I mean the number of patients discharged as cured or as socially recovered (because the staff is particular in the use of the word "cured"), is large. I know of no institution of its size which discharges as many patients a year as cured or as socially recovered as does the Hastings Hillside Hospital. I will repeat what Dr. Glueck said: "The success of this method lies not only in selecting the proper persons to receive treatment, but particularly in the therapeutist, and Dr. Wender, himself, is the master of therapy." We realize that in treating a group like this there is great danger inherent in the method. One must see that these patients are not psychologically conditioned. Much of the lecturing to the public carries a danger that the listeners may become impregnated with deleterious ideas concerning mental disturbance. They acquire an attitude of fear of the normal. They become introverted. That is the danger in the method, as Dr. Wender pointed out, which, through his experience, he is able to avoid.

The actual happenings at these sessions are occasionally taken down stenographically, and it is interesting to note that the patients criticize each other. At one session a patient made a statement regarding an occurrence in her life and gave her interpretation of it. Another patient objected, remarking that the explanation given was superficial and that there must be some deeper underlying fact which that patient had suppressed. In other words, they become their own therapeutists. They also learn that mental illness is not exceptional; that many people have such illness and there is no stigma attached to it. I want to stress that point. It has been the endeavor of the hospital, and it is the endeavor of every psychiatrist, to educate the public to regard mental disease as a disease on the same level as any bodily disease; to show that it carries no shame, and that a patient is just as good as the next man when he has recovered. He has merely been unfortunate in becoming mentally ill.

The patients have formed what is known as the Wender Welfare League, which meets regularly. At their second meeting 70 were present. One of them said: "Before I entered the Hastings Hillside Hospital I had friends. Since I left there I no longer have the same friends, but I have other friends that I met there, and they are better friends than any I have ever had."

I wish to add that all patients in the hospital do not submit to this form of therapy, and naturally other forms are undertaken. As Dr. Brill said, it is almost impossible in any hospital of any size to give individual attention to every patient. However, if one can in any way make a short cut which leads to the reestablishment of a patient's integrity and socialization, one possesses something which promises a great deal in future treatment in hospitals, provided it is used in a proper manner.

DR. BERNARD SACHS: This matter of group psychotherapy strikes me as sensible, and I confess that I feel a little as Dr. Diller expressed it, that it is not new. In fact, it occurred to me while it was being discussed that the matter of group activities is well known in many different directions. The school system

is based largely on group activities, and all know that the association of children with one another enables them to learn their lessons much better than if they were treated as individuals. At the same time, I hope, in spite of the success of the group psychotherapy method, that the treatment of the individual will not be neglected altogether. I cannot help thinking that this is one of the sore spots in sanatoriums; there is too little individual treatment, and if there is individual treatment it is often, to my mind, of the wrong kind. I am glad to know that this group system is on the whole a rather rational one. It is a pleasant surprise to me. I do, however, want to add: I was wondering, as Dr. Wender explained his method, as to the wisdom of those "talks." Does he not put a lot of ideas into the heads of these patients, and then when later he tries a sort of free association method on the patients is he not going to have them say what he put into their heads in that beautiful free association method? Perhaps I understood Dr. Wender incorrectly and am mistaken about this, but my idea was that he gave them a lot of psychic theories and put a lot of ideas into their heads which they are not benefited by having; if anything, one makes introverts of them by just this method. I have had a chance to read the reports of the Hastings Hillside Hospital. It is encouraging to know that the matter is being handled there as well as it is, and I hope the group psychotherapy method will prove even a greater success than it has, but let me put in a plea for rational individual treatment as well.

Dr. Clarence P. Oberndorf: What struck me most in the paper of Dr. Wender was the mechanism through which therapy operates in such a group. I think that it is much along the lines of Coué psychotherapy, namely, as in the Coué method, the cure is put in the patient's own hands and the patient becomes his own doctor. So, in this method through identification with Dr. Wender the patient becomes the doctor and cures himself.

While all that has been said regarding the value of the interaction of a group of persons on one another is true, I think that it is particularly worthy of emphasis that such patients prefer to be in a group of ill persons rather than in a group in which the majority are normal. We have had that experience at the Mount Sinai Hospital in an outpatient social club during the past nine years. This club is made up of persons, so to speak, who have not necessarily graduated, but who have discontinued coming to the clinic. The patients come back regularly to evening social meetings and feel that they have the support of each other, that is, that they belong to a group. Through identification with the leader, a skilled occupational therapeutist, they feel a certain power to cure themselves.

Dr. Louis Wender: In answer to Dr. Sachs, during the course in group psychotherapy, or following it in individual interviews, very little free association is practiced. Everything is on a conscious or preconscious level; therefore, it is felt that what the patient learns through the lecture material has no effect whatever on the conscious material he produces. As a fact, it is felt that it stimulates the release of unconscious ideas.

As to Dr. Diller's statement concerning Dr. Rush's work, I regret to state that I have not read his "Textbook in Psychiatry."

PHILADELPHIA PSYCHIATRIC SOCIETY

Regular Meeting, April 12, 1935

Joseph C. Yaskin, M.D., *President, in the Chair*

A Possible Case of Pick's Disease. Dr. James Greenwood Jr.

The patient whom I am presenting shows many of the features of Pick's disease. The onset of symptoms, which was sudden, very much in the manner of a vascular

accident, would not explain the rather severe degree of cortical atrophy shown in the encephalogram. A vascular origin is also rendered a little unlikely since there is no evidence of either retinal or radial arteriosclerosis.

J. J., a Negro, aged 36, was admitted to the Philadelphia General Hospital on Aug. 17, 1934, with the chief complaint that he was unable to talk properly and was acting in a queer manner. The history was somewhat confusing in that it was claimed that he was perfectly normal until the morning of admission, when he suddenly lost the power of speech, did not recognize his relatives and was unable to walk. A more detailed story showed that he lost his job four or five years before and shortly thereafter began to use alcohol, chiefly in the form of ale, and was unable to obtain further employment. Under the present economic situation it would not be fair to say definitely that he suffered a change of character. On the night before the illness supposedly began he drank a little more than usual, although his wife states that he never became intoxicated to an objectionable degree. On the following morning he awoke, felt ill and nauseated and experienced a severe attack of vomiting. Following this he appeared confused, and was unable to talk except in a disconnected manner but was able to walk.

Examination revealed a rather confused and disoriented man who showed in addition to the changes in the sensorium definite aphasia of the sensory type. He could name only about one-half the objects shown to him and was unable to execute any but the most simple commands (he was unable to obey the request to put his right hand on his left knee but put it instead on his shoulder). He was able to read a written command aloud but was unable to carry it out. Neurologic examination gave negative results—the pupils reacted to light, the eyegrounds were normal and there was no retinal arteriosclerosis. There was no facial weakness, and the strength was equal in the two arms. All the deep reflexes were slightly exaggerated, and there was a slight tremor of the hands and tongue. In the absence of any vascular changes a tentative diagnosis of tumor of the left temporal or parietal lobe was considered. A toxic psychosis, possibly due to alcohol, was also thought of. A vascular accident, of course, could not be ruled out. Physically the patient appeared to be healthy, and close examination revealed nothing except subacute tonsillitis. The blood pressure was 120 systolic and 75 diastolic. Laboratory studies gave essentially negative results; these included chemical examination of the blood, urinalysis, a blood count and a Wassermann test and also a provocative Wassermann test of the blood and spinal fluid.

An encephalogram gave more information. The amount of fluid removed, 165 cc., was appreciably higher than the average (110 cc.) and was not by any means all that could have been obtained; 160 cc. of air was injected. Roentgen examination showed a rather extreme degree of cortical atrophy, particularly well visualized in the frontal and parieto-occipital regions. In view of this finding, my colleagues and I were unable to reconcile ourselves to the belief that all the symptoms had come on suddenly. Whether the precipitation of the illness came as a result of a vascular accident or as the result of a toxic process due to alcohol or tonsillitis it was not possible to say; however, the picture was one of dementia complicated by aphasia, and in the presence of cortical atrophy it was thought that he had some form of presenile dementia, most likely the type described by Pick.

During the patient's stay in the hospital there was slight improvement. He became fairly well oriented but had some difficulty in expressing himself and still appeared confused. He was active at times and agreeable and attempted to cooperate. He was discharged to the Philadelphia Hospital for Mental Diseases on Oct. 10, 1934, and paroled from there on October 26, as somewhat improved. A recent follow-up study shows essentially the original findings, slight dementia, a slight sensory type of aphasia and the appearance of mental alertness which is more apparent than real.

DISCUSSION

DR. SAMUEL ZERITSKY (by invitation): I examined this patient on the day of his admission to the Philadelphia Hospital for Mental Diseases, and at that

time he was not aphasic. He was entirely disoriented, and his memory was poor for recent events. There was no palsy, but the history of sudden onset and inability to walk at the beginning of the attack was definite. So far as the cortical atrophy is concerned, one must accept it as present on the basis of the roentgen report, but I think it will be necessary to watch the patient further before the diagnosis can be accepted.

A Juvenile Psychopathologic Study. Dr. Frederic Leavitt.

D. S. C., aged 8 years and 8 months, a Jewess, is presented because she has shown, since the age of 2 years, definite psychopathologic traits that tend to place her in the schizophrenic group at this early age. Her problem is essentially in relation to peculiar antipathies, fears and obsessions, an exaggerated fantasy and day-dreaming state and an extreme degree of psychomotor and physical hyperactivity, all of which have contributed to the development of a markedly introspective type of personality, practically to the point of the development of secondary personality types. On cursory examination several years ago it was believed that the child was merely mentally defective, but during the years she has been under observation at the Devereux School an interesting series of psychopathologic reactions have been observed which place her more in the "constitutional psychopathic" defective class, with a marked tendency toward the development of schizophrenia. An interesting feature is a "withdrawal" phenomenon, by means of which she avoids an adverse environment; especially an "alteration of personality" in which she becomes "Miss Stone" for a period sometimes of days, and during which she reacts as an entirely different person.

The family history reveals no instances of mental disease or defect. The patient is the youngest of three children, two brothers, aged 15 and 10 years, being normal in all respects. The patient was born at term, talked at 10 months, had no difficulty in articulation, but did not walk until 25 months of age. During the first two years of her life the parents noticed that the child took little interest in objects about her and did not acquire as large a vocabulary as most children do in that time. There is no history of any serious illness or accident during the preschool years. She was sent to a private kindergarten, where it was observed that she was asocial in her behavior.

Between the ages of 2 and 5, her father, not understanding her, gave her corporal punishment frequently, which may account in part for her "fear reactions" and her "withdrawal" tendencies.

Dr. Edward M. Westburgh and Miss Helen Brace supplied the following psychologic reports. By the Stanford revision of the Binet-Simon test in January 1933, the intelligence quotient was rated 78. The examiner believed that the patient had subaverage intelligence, but it did not function because of her psychopathic personality. In several tests she failed because of inability to keep attention on the matter in hand long enough to comprehend what was wanted. She showed lack of interest and concentration and marked distractibility, and she insisted on using the test material to suit her own desires rather than obey the wishes of the teacher. At that time the patient was so interested in her play life, particularly with make-believe dolls, that it was extremely difficult to make her focus attention on her surrounding environment. Each time a request or question was given, attention had to be gained anew. To tests which she particularly disliked she reacted with screams of remonstration. When she was permitted to use the test material in her own way, she responded with screams of delight.

The second Binet-Simon test was made in March 1934, when the intelligence quotient was rated as 85. At this time it was noticed that the child's concentration was better, but it was only fair as regards her ability to grasp and remember material such as digits and sentences. Her ability to carry out the test was greatly interfered with because of great emotional instability and a constant fantasy state, in which she manifested a decided preference to live in a realm of unreality rather than face actual situations which were at times unpleasant to her. Owing to the training she had received at the school during the fifteen months

following the first examination, it was noticed that she displayed better ability in handling concrete material, and that she showed evidence of becoming interested in school associates and other persons and in things of her contemporaneous environment. It was noticed that anything the patient liked to do was performed exceedingly well, but when she was forced to do tasks that were unpleasant to her, the reaction was one of resisting "negativism."

A third psychometric examination was made on Feb. 13, 1935, by which the intelligence quotient was rated as 70.

The record of behavior and habits is that characterizing a personality dominated by fantasy and day-dreaming, marked seclusiveness and a tendency to withdraw from reality. The child is never so happy as when indulging in play with make-believe people, often with herself as an entirely different personality, and to this "alter ego" she frequently gives another name. She is extremely hyperactive, detests rest periods and is always noisy during these periods. When she first went to the school she would not play at all with other children; it was impossible to get her in group activities. Once in a while she would play a game if it happened to suit her fancy, but she much preferred going off by herself in a corner and, using blocks, playing active and ingenious games. She has a very vivid imagination, which so absorbs her that she seems actually to "live" the play in which she indulges. She inclines to be interested much more in pretending than in actuality. She pretends tea parties and going to movies and lives over in her play past experiences she has particularly liked. It has also been noted that if an actual situation does not please her she tends to withdraw from the actuality of that situation by reverting to her realm of unreality by taking unto herself other names, the most frequent of which is "Miss Stone." When she is "Miss Stone" she seems to assume an entirely different personality and apparently does not remember any of her reading vocabulary or how to print or write; her voice, actions and mannerisms are very different at those times, but she does not drop out of doing her usual routine things. As "Miss Stone" she exemplifies the personality that is distasteful to her normal personality. When she meets a situation that she cannot cope with or one that is unpleasant or distasteful to her she becomes "Miss Stone" as a buffer personality to protect that of the real D.S.C. She has frequently said: "I am like two people—Miss Stone and D.C. When will Miss Stone go away, and if she does go away will I see her anymore?" At other times the patient apparently knows that "Miss Stone" exists only in her mind. A few years ago "Miss Stone" would be in the ascendency for days at a time, but following three years of training at the school this second personality type has been present very little, generally only for a matter of minutes or, at most, an hour or so. "Miss Stone" seems to embody projection, identification and a refuge mechanism to the patient. She is a "make-believe" person in her mind on whom is projected all the horrid and terrible qualities D.C. can think of. At the same time, D.C. definitely feels that "Miss Stone" is identified with her and is a real part of her make-up.

At times, in her fantasy plays, the stair newel posts and the Indian clubs in the gymnasium represent real play companions to her in her games. She will stand in front of the newel post and talk to it as a second person.

This girl is also subject to numerous psychasthenic phobias which affect her emotionally. She is hypersensitive about food and will always smell a new food before she will attempt to eat it. For a time she had a very marked fear of dogs, and the "unknown and unacquainted" were terrifying to her. She had a marked fear of closed doors; when she first went to the school she could not bear to see a door close but would immediately run to open it and protest violently if further attempts were made to shut it. By now this fear has been entirely eradicated. The night terrors, which were so frequent and disturbing when she first went to the school and which she stated resulted from vivid dreams, are no longer present. Her former fear of dogs, closets, closed places, darkness, "spectacles" and being hurt do not, at present, dominate her life. For a time, moving swings, tricycles and water caused her great emotional distress, but these fears have been dissipated.

She still fears corporal punishment or the possibility of being hurt, and when the prospect of such is in the offing she still demonstrates a tendency to assume the "Miss Stone" personality, who represents to her the "bad part" of herself. She is still not quite sure that she really is one person, "D.C.," and she still believes that she may be several persons and can change into any one of them at will. It has also been noticed that if another child in the school is praised for something he or she has done, the patient will frequently take on herself the name of the child being praised. This, however, is not a constant tendency. She is curious about anything that takes her attention and will go to great extremes to investigate it thoroughly and if necessary pull it apart to do so. Many of her obsessions are connected with things she fears or which she does not understand.

She apparently has considerable sex curiosity, and the method of teaching has been to tell her the truth in every instance. With this procedure she seems to have been satisfied, and at the present time there is little along sex lines that she is disturbed about, except that she apparently takes delight in suddenly speaking certain words to strangers and observing how they react, particularly if in a shocked manner. She has a few choice words which she uses in relation to sex which appear to her to be "shocking," and they apparently have some relation to a sex episode that occurred when she was between 2 and 3 years of age and which roused in her a great deal of excitement and horror.

When the patient first went to the school she was almost totally inaccessible and evidenced a desire to live entirely a fantasy life and consciously and subconsciously endeavored to put reality out of her life. In relation to this, she was beset by fears and obsessions, intense jealousies, repressions and regressions. On her first day at the school she almost demolished the office. She jumped around on her toes continually, grabbing at first this and then that, throwing the articles down, laughing wildly one time and giving piercing shrieks the next minute. When she found that her father would not snatch her and slap her as she expected him to do, she grew much worse in her misbehavior. There then ensued some months of the most hysterical kind of behavior—screaming, laughing, trying to kick, hit and bite, refusing to eat properly, not sleeping at night and being a general disturbance to the entire household. She was too disturbed to be taken to a physician's office. She was never punished in any way, and every one handling her kept calm and serene no matter what she did. She seemed to get worse from day to day, descending to an almost vegetative level, where she soiled her bed and clothes. Then she began to talk more and gradually wanted to join in the children's play.

The most difficult obsession to overcome was the desire to make dolls and her aversion to real ones. She showed great ingenuity in taking rulers, blocks, cushions and anything at hand which could be wrapped in a coat, dress, apron or even a rug from the floor to make a doll. She invariably arranged it so that she could pull the thing that represented an arm off, and then she would scream and dance, yelling "the baby's dead" or "baby's killed," and refuse to be comforted. At first an effort was made to put back the arm, but that increased her yelling. Then the whole thing was cleared away, and she would go into a temper tantrum for hours. Finally, it was decided to keep her from making these dolls. This worried her, and her whole day was spent trying to struggle to find things to make them. The device was arranged to allow her to draw dolls, and she showed plainly that the arm represented a sex organ, and from that time she began to talk so that she could be reached. By constant association with other children in the school and by a method of training she is emerging more and more from the "shut-in" type. She is much more spontaneous in conversation, but is still highly distractible. It is noted that if she could choose her own procedures in a psychometric test, she would qualify with a relatively high intelligence quotient, but these few procedures are counterbalanced by the great number to which she has antipathies, which pull down her average greatly.

The child exhibits a typical leptosome type of physical development. She is 4 feet and 5 inches tall (134.6 cm.) and weighs 59 pounds (26.8 Kg.) with ordinary

clothing on. She presents numerous anomalies of development, the most noticeable of which are the extremely long, tapering fingers and toes, the exaggerated length of the long bones, a tendency to extreme flushing and dermographia of the skin and constant psychomotor and physical activity. There is extreme hyperflexibility of all joints of the body. Although she is 6 pounds (2.7 Kg.) underweight, she is reported as healthy and resistive to infection; it is apparent that she burns up fat rather than carries it. The pulse is rapid, ranging around 120; after exercise of hopping up and down for one minute the pulse rate increases to 140, but with a continuance of good tone and rhythm. Otherwise physical examination reveals no abnormalities. Examination of the central nervous system reveals no anomalies or pathologic conditions.

Summary.—This patient presents a quantitative mental defect which appears to be accentuated in great part by a psychopathic personality reaction of the extreme introverted or "schizoid" type. The progress that has been made at the school to bring her more in a state of awareness with her environment has shown decided results, and it is possible that by this means she may be saved from what was her predestined end—schizophrenia.

DISCUSSION

DR. CHARLES W. BURR: I do not like to praise this twentieth century, but it has seen much of value in the study of supposedly diseased children. When I was young no attention was paid to these persons. They were called feeble-minded and disregarded. I also must praise Freud, although the members know what I think of his theory. He has done more, I think, than any man in the past thirty years to stir up interest in studying abnormalities in growing children. This child has not had any test of intelligence at all. I am convinced that many intelligent children, according to these tests, rate very low. The real reason that they rate so low is that they are so interested in things of their own fantastic world that they do not take seriously the problems put to them in the Binet-Simon test or any modification of it. I do not think that it was feeblemindedness which made this child pass so poorly in the mental test. I think that she was saying "What nonsense this is! I want to think about, or be, Miss Stone." That is one of the things that decreases the value of mental tests.

Many years ago a friend who had two children said to me: "Dr. Burr, I am afraid my boy, Tom, is going to grow up a liar. Tom all the time persists in playing with something that does not exist." Tom was 7 years of age. Tom had a friend. They lived up in the northern part of town, and his sleeping room was on the second floor looking out on the main street. This boy of 7 had developed a change in his own personality. When questioned and requestioned, directly and indirectly, it was never "Dr. Burr, I am playing," it was "Dr. Burr, Borlax lives down on the cobblestones. Whenever I am alone I call him and he comes and plays." That was the only mental symptom that the boy presented. He grew up an ordinary, everyday man, neither very low in mentality nor very high. He was fairly successful, a college man, and finally became a minister. His brother, who showed nothing abnormal in his childhood, who never had imagination enough to make up friends, who did exceptionally well at school and who passed splendidly at college and then studied medicine, took to drink and was under my care in an institution for mental disease.

DR. MILTON K. MEYER: I wish to ask Dr. Leavitt whether there were any abnormalities in personality before the sex trauma. It seems that the sex episode might have something to do with the mental condition. While the record states that the child's neck was thin and the thyroid gland was not palpable, it looked to me as if the thyroid gland might be slightly palpable at times. If a basal metabolic test has not already been made, one may reveal some disturbance of the thyroid gland. The pulse rate is high. This case reminds me of one I saw many years ago in which full-fledged exophthalmic goiter was present in a girl about this age.

Dr. Frederic H. Leavitt: I have been unable to obtain any answer for the first part of Dr. Meyer's question as to what the child's psychopathologic condition was prior to the sexual trauma. I think that the sex episode may have had some influence on her condition. I do not look on the case as one of thyrotoxicosis. There is constant tachycardia, 120 most of the time. The patient does not present the usual symptoms accompanying this disease. Her condition is rather functional hyperthyroidism.

Atrophy of the Optic Nerve in Dementia Paralytica: Its Relation to Tryparsamide Therapy. Dr. Robert S. Bookhammer.

In the optic tract, functional loss precedes morphologic change. Consequently subjective signs of visual failure occur before atrophy of the optic nerve can be demonstrated objectively. The most important symptoms are dimness of vision and blurring and flashes of light. There is a contraction in the visual fields, and later there are changes in the fundi.

The object of this report is to record the experiences in the men's department of the Philadelphia Hospital for Mental Diseases during 1932 and 1933 with tryparsamide and its relation to atrophy of the optic nerve. The study was made on a group of 262 patients whose eyegrounds were studied under the supervision of the consulting ophthalmologist, Dr. Max Gabrio. The visual fields were not determined because the mental condition of most of the patients would not permit such a procedure. Of these patients, normal eyegrounds were observed in 183, or 69.6 per cent. Fifty-three, or 20.2 per cent, were found to have atrophy of the optic nerve prior to treatment. Of these, 10, or 3.8 per cent, had well advanced atrophy of the optic nerve; 9, or 3.4 per cent, had moderately advanced atrophy, and 34, or 12.9 per cent, had incipient atrophy. Twenty-six, or 9.9 per cent, of the 262 patients had eyegrounds in which there was some doubt as to whether or not tryparsamide should be employed. While in these cases there was no evidence of atrophy of the optic nerve, the disks were slightly off color. In this group it was decided to use tryparsamide with caution and to recheck the condition of the eyegrounds at frequent intervals. Nine, or 34.6 per cent, of these patients presented subjective and objective signs of atrophy of the optic nerve. Of the 183 patients whose eyegrounds were considered normal, 10, or 5.4 per cent, acquired atrophy of the optic nerve. The average number of doses of tryparsamide given before the ocular changes were noted was seven. In 1 patient changes developed after the first dose; another patient had twenty-two injections of the drug before visual disturbance was noted.

Judging from the experiences cited, it seems that atrophy of the optic nerve in dementia paralytica depends on the presence of syphilitic disease in the optic tract. In this group, tryparsamide does not appear to have any selective affinity for this tract but may act indirectly on the optic nerve by stimulating the latent pathologic condition present. No one should attempt to treat neurosyphilis with tryparsamide without having thorough ophthalmologic examinations made prior to and during treatment. This examination should include, if possible, a test of the visual acuity, determination of the visual fields and examination of the fundi. In patients in whom the disease has advanced so far that they require care in a hospital for mental diseases, examination of the fundi is the only reliable means to determine whether or not changes have taken place, because the psychotic state of most of the patients precludes the possibility of accurate subjective observation. Some observers have reported that tryparsamide in some cases actually improves the condition of the optic nerve in the presence of atrophy. No comment concerning this can be made because other therapeutic agents have been used when atrophy of the optic nerve has been recognized.

DISCUSSION

Dr. Samuel Hadden: Tryparsamide was introduced to the medical profession with a known bad heredity because of its derivation from sodium arsanilate, which was known frequently to produce atrophy of the optic nerve. When its use was

first begun at the Philadelphia General Hospital it was requested that special studies of the visual fields and eyegrounds be made in all cases as a means of preventing the occurrence of atrophy of the optic nerve. Routine examinations of the visual fields were soon discarded because as a rule they were made worthless by the degree of deterioration present in most cases. Greater reliance was placed on studies of the eyegrounds and a carefully obtained history to select the cases in which the drug seemed to be contraindicated.

At present my associates and I believe that the important contraindications to the use of tryparsamide are not generally recognized by many ophthalmologists. Almost invariably they advise against the drug in the presence of retinochoroiditis, but in a group of patients having this condition and treated with tryparsamide no visual catastrophies were experienced.

We believe that the most important contraindications are:

1. Any degree of atrophy of the optic nerve. However, if after a period of observation and from a carefully obtained history there is no tendency to progress, the drug is given cautiously, and we believe that more of these patients have been benefited than harmed.

2. A history of subjective visual disturbance, such as night blindness, scintillating scotomas or reading difficulty of recent onset.

3. The slightest evidence of neuritis of the optic nerve with filling of the disks or fluffing of the margins of the disks.

From experience, we recognize that this last group is the one in which there is the greatest danger of amblyopia developing when tryparsamide is given. We believe that with reasonable attention to those abnormalities there is no greater danger of atrophy of the optic nerve developing when tryparsamide is used than when other arsenicals are used. We believe that we can fairly predict the occurrence of atrophy of the optic nerve by observing these details. The last three cases of atrophy of the optic nerve observed in our clinic were anticipated and other methods of treatment were used which are accepted as safe as far as the danger to the vision is concerned. In all cases routine studies of the eyegrounds are made. In those in which the drug is considered to be contraindicated by the ophthalmologist we reexamine the eyes, and unless one or more of these conditions is found to be present we give the drug. Far more patients are helped than are harmed.

PSYCHOTIC REACTIONS ASSOCIATED WITH HYPOGLYCEMIA OR AN ARTIFICIALLY INDUCED HYPERINSULINISM WITH DIFFERENTIAL DIAGNOSIS BETWEEN THESE AND SCHIZOPHRENIA. DR. EDWARD B. ALLEN (by invitation).

Two cases were reported of hypo-insulinism induced by insulin therapy in the treatment of diabetes. In one case it was acute and in the other chronic. Both mental and physical symptoms were noted. In the case of chronic hyperinsulinism the mental symptoms were differentiated from those of schizophrenia. The following conclusions were drawn: In distinguishing between hypoglycemic reactions and schizophrenia from a study of the psychotic reactions one must keep in mind the distinction between organic and functional psychiatric reactions. If one understands this distinction, the differentiation is much easier. The important thing in hypoglycemic reactions is the disturbance of memory and retrospective amnesia for what has transpired during the reaction. In schizophrenia there is no impairment of perception, memory or intelligence, and one should look for the underlying primary disturbances of association and affect, which, according to Bleuler, are identical but seen from different angles.

DISCUSSION

DR. EDWARD S. DILLON (by invitation): Dr. Allen has touched on so many interesting points that it will be possible for me to discuss only a few of them. In treating patients for diabetes mellitus one has the following aims: (1) to prescribe a diet properly distributed in protein, fat and carbohydrate and containing suffi-

cient calories so that the patient will be neither overnourished nor undernourished, and (2) either without insulin or with it to keep the blood sugar level below the renal threshold and above the level at which shock is likely to occur. For the majority of patients, after they have learned their diets accurately and have been properly standardized this is not particularly difficult. In some patients, however, the blood sugar level is unstable and changes rapidly in a few hours. For these patients the size and distribution of the doses of insulin must be gaged with great accuracy, otherwise the blood sugar will be too high at some times of the day and too low at other times. These patients are likely to have frequent insulin reactions.

During the past ten years there have been approximately 3,500 diabetic patients in the wards of the metabolic department at the Philadelphia General Hospital. Most of the patients have had severe or moderately severe diabetes. For some patients in the wards to have an insulin reaction is an almost daily occurrence. There are 13 graduate nurses in the department, who are specially trained, and they must be on the alert to detect and treat reactions before they are severe. The nurse always gives the patient orange juice wthout calling a physician and without waiting for the report on the blood sugar, blood always being taken at the time a reaction is suspected so that one may know the blood sugar as a matter of record. If a patient previously has been mentally clear, the fact that he has become confused immediately calls attention to him. Many of the patients, however, are already confused from some other condition, such as advanced heart disease, a serious infection or senility. Often the nurse's attention is attracted to a patient by a change in his emotional state or by his cold clammy skin. Weak patients must be watched with great care for oncoming insulin reactions, as signs are not easy to detect. The symptoms of a mild to moderate insulin reaction are that the patient feels weak, nervous or hungry, breaks out in perspiration and is mentally confused in the sense that it is difficult for him to concentrate. His emotional state may become unstable. Women may cry, and men usually become hilarious. Often a reaction will begin while the patient is reading, and the first symptom will be that the words run together. Patients can nearly always feel symptoms coming on, and if orange juice or sugar is taken the symptoms pass off in the course of from five to fifteen minutes. In an occasional patient the symptoms come so quickly or the patient's attention has been so engrossed in something that by the time he is aware of them he has lost the ability to act. The reaction is then likely to be severe unless he is rescued by some one else. In a severe reaction the patient usually merely gradually sinks into profound unconsciousness. Occasionally there may be convulsions.

What are the dangers of insulin reactions? They are chiefly two. These dangers are: First, that something will happen to the patient in the way of a traumatic accident. He may be on the street and get in the way of an automobile or street car, or he may fall down stairs or injure himself in some other way. The second great danger is that some one will mistake the reaction for diabetic coma and give the patient more insulin. Unfortunately physicians often do this. It is not difficult to make a differential diagnosis between the coma of diabetic acidosis and that of a severe insulin reaction. It is very difficult, however, when a diabetic patient is unconscious and in acidosis to tell whether he is unconscious from some other condition, such as apoplexy, an attack of heart disease, alcoholism or severe toxemia. Other dangers described in the literature are badly overrated. It certainly is inadvisable for an elderly arteriosclerotic patient to have too low a blood sugar content. The literature contains reports of a number of cases of "heart accidents" due to a low blood sugar content, but such a case has not been seen at the Philadelphia General Hospital. There have been 73 deaths from heart failure in the last four years. Not one of the 73 patients had a low blood sugar content at the time of death. A check up is always made by making a test for sugar on blood from the heart immediately at death.

Various neurologic complications due to hypoglycemia are mentioned as being fairly frequent, for instance, hemiplegia. I have seen only 1 case. The patient had hemiplegia without any mental confusion. He took orange juice, and in the

course of an hour the hemiplegia was entirely gone. At one time he was severely diabetic and had taken insulin for eleven years, but at the time of the hemiplegia he was taking 15 units a day. This was cut in half, and he had the same reaction a week later. There was complete hemiplegia on the left side, which passed away after the ingestion of orange juice. Following this he received no insulin for eight months, but two months ago he had to resume the use of insulin because the blood sugar level became high.

As for gastro-intestinal complications, I do not think they exist because of hypoglycemia. When one has an upset stomach, however, one is likely to have an insulin reaction, because carbohydrates are not absorbed. Too little food is just as bad as too much insulin.

Only 1 patient has died of hypoglycemia at the hospital. This patient, Joe, liked to go off on sprees. He had fairly severe diabetes. Several times he went off on a spree and had to be admitted to the hospital because of coma. On this occasion he came home in time for supper one night, took insulin and then, owing to the alcoholism, vomited his supper. About 9 o'clock the family thought he was going into coma. They gave him a syringeful of insulin. About an hour later Joe was worse, and they gave him another syringeful of insulin and then went to bed. In the morning Joe was found unconscious, and his blood sugar was 22 mg. per hundred cubic centimeters when he was admitted to the hospital. He was given dextrose intravenously but died four hours later without regaining consciousness. Usually a patient with severe hypoglycemia recovers promptly after the intravenous administration of dextrose.

Is the memory of what happens during an insulin shock impaired? This varies. A patient crossing Independence Square felt an insulin shock coming on but did not eat sugar. A policeman noticed that he was acting queerly. The patient told the policeman to "go to hell." The policeman then realized something was wrong and took him to a hospital, where he was unconscious for over an hour without a diagnosis being made. As soon as the physicians were told that the man had diabetes he was given dextrose intravenously and promptly recovered. He remembered telling the policeman to "go to hell," but nothing later. Another time a physician called me and said that a patient was in diabetic coma. He had given him 20 units of insulin, and on going back two hours later he found that the patient was worse. I sent an ambulance, having first advised the physician not to give any more insulin as he gave very distinct symptoms of an insulin reaction. The patient arrived at the hospital before me. The chief resident recognized the condition and gave dextrose intravenously. When I arrived I found the patient, aged 22, sitting up in bed with his family and the priest standing by the bed and much astonished at the sudden recovery. He said that he remembered when the physician came and gave him the insulin and that he knew that was the wrong thing to do. He remembered also not being able to swallow orange juice. Another patient, however, who worked in New York and lived in Harrisburg, left for home one night on a 6 o'clock train, and while he was on the train a severe insulin reaction developed. Three times he pulled the bell cord. The porter took him into the baggage car; the first thing he knew, he woke up in the baggage car in Altoona at 2 a. m. and remembered nothing of his experiences on the train.

In regard to Dr. Allen's case: The patient was unconscious for eight days. She could not have had hypoglycemia for eight days. I am talking from the point of view of chemistry, not of psychiatry. Even though one does nothing at all for such a patient, sooner or later (usually in eight or ten hours) he will recover. Metabolic processes are going on just the same, and even if food is not given carbohydrates will be withdrawn from his own body. An injection of insulin has effect for about eight hours.

The symptoms which accompany hypoglycemia, whether due to hyperinsulinism or to some other dysfunction of the endocrine system, are so almost exclusively referable to the nervous system that the psychiatrist is sure to encounter these patients before the metabolist, and many will require the services of both the psychiatrist and the metabolist to obtain the best diagnosis and treatment.

DISCUSSION

DR. R. A. MATTHEWS: Has Dr. Allen studied any cases of spontaneous hyperinsulinism?

DR. EDWARD B. ALLEN: I am certainly grateful to Dr. Dillon for his discussion of my paper and for bringing out the physical signs of this condition in the lucid and thorough manner that he did. He spoke about the patient being unconscious for eight days and also being hypoglycemic for that period. I am fully in accord with that. The father claimed that the patient had been unconscious for eight days but discovered that by mistake sugar had been given. The blood sugar rose from 30 to 400 mg. per hundred cubic centimeters. During this period the patient was unconscious. I rather question whether the patient was unconscious for eight days. I was interested in what Dr. Dillon said about the fact that sometimes a patient remembers what had transpired at the beginning of the insulin reaction. This depends somewhat on the severity of the reaction and also on the time of the reaction. If he is going into the reaction he may remember something that happened at that time.

As regards spontaneous hyperinsulinism, I have had no such cases, but have known about them and am on the look-out for them.

WEIGHT FACTORS IN PSYCHONEUROSIS. DR. W. L. LONG.

This study presents an analysis of the weights in histories of 94 psychoneurotic patients. The group was selected from over 1,000 office records as representing a typical clinical picture of psychoneurosis. Consideration is given to the weights of patients from the time of the sickness until recovery. I have compared the weights at the time when the patients were first seen with the standard weights for their age and height, with their maximum weights before treatment within three years, with their minimum weights before treatment within three years and with their weights after treatment was completed. There are tables showing the incidence of the more common symptoms. The results of the treatment are classified as either recovery, improvement or failure. Of the 94 patients studied, 39 (41.4 per cent) were men and 55 (59.6 per cent) women; 49 (52.5 per cent) recovered and the condition of 27 (29.1 per cent) improved after treatment; 18 (18.4 per cent) received no benefits. Twenty-one (53 per cent) of the men recovered and 28 (59.1 per cent) of the women.

The decrease in weight from the patient's maximum within three years was plotted against the number of patients. It shows graphically that the majority of patients show considerable loss of weight before becoming ill. It is interesting to note that of the 94 patients 76 had weighed more than their weight on admission at some time previously. The sequence of loss of weight and development of symptoms occurs repeatedly throughout this series.

Twenty-four selected patients were divided into two groups, as follows: Group A, consisting of persons in whom I believe spectacular results were secured, and group B, consisting of patients in whom we considered the results of treatment a complete failure at the time of their discharge from the hospital or when they left our care and also of those whose condition was either unimproved or worse at the end of the treatment. Of the patients in group A, 11 gained weight, and of those in group B, 3 gained weight. Here it is strikingly shown that inability to gain weight is a poor prognostic sign. Of the 3 who gained weight in the group in which treatment was of no benefit, 1 increased in weight from 70 to 90 pounds (31.8 to 40.8 Kg.) and should have weighed 125 (56.7 Kg.), and one gained 35 pounds (15.9 Kg.) but probably did so because of dysfunction of the pituitary gland.

DISCUSSION

DR. HAROLD D. PALMER: How many psychoneurotic patients who were overweight were reduced at the start of treatment?

DR. W. L. LONG: I don't think that I have reduced the weight of any of them, but I have kept most of them at their upper level until the psychoneurosis was controlled.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

C. A. McDonald, M.D., *Presiding*

April 25, 1935

Meningitis Due to Streptococcus Haemolyticus: A Proved Case, with Recovery.* Dr. Robert S. Schwab.

E. P., a Canadian housewife, aged 44, entered the Massachusetts Eye and Ear Infirmary on Feb. 27, 1935, with a history of a discharge from the ears for five days. Except for a simple mastoidectomy on the left side ten years previously, the past, family and marital histories were irrelevant. The patient was entirely well and at work until two and one-half weeks before admission to the infirmary when she caught cold and had a severe sore throat with a temperature of 102 F. Six days before admission the left ear was painful, and there was a discharge; the following day the right ear was so sore that a physician had to lance it. Three days after the onset of the otitis media there were stiff neck, transient diplopia to the right and fever and chills.

On admission to the hospital, in addition to the discharge from the ears, there were a red throat, stiff neck, râles at the base of the right lung and a temperature of 103 F. The urine contained many white cells, and the Benedict test for sugar gave a green precipitation. There were 15,000 white cells in the blood. The patient was alert, clear mentally and not toxic. In the absence of Kernig's sign, the neurologic consultant postponed lumbar puncture, and the patient was transferred the next day to the East Medical Service at the Massachusetts General Hospital. During the next six days the râles spread to the base of the left lung, the condition of the throat improved and a diagnosis of diabetes was ruled out. Fever persisted, together with the stiff neck, and on the seventh day because of the absence of the knee jerk on the right and the presence of a suggestive Kernig sign, the first lumbar puncture was done. It showed a pressure of 500 mm. of water and partial dynamic block. The fluid contained 1,000 polymorphonuclear cells and had a sugar content of 9 mg. per hundred cubic centimeters.

The patient was transferred to the Neurologic Service, where a combined lumbar and cisternal puncture was performed immediately. This showed complete block, and the specimens of fluid were essentially the same except that the sugar content of the fluid obtained on cisternal puncture was 41 mg. Culture of all three samples of fluid showed an abundant growth of Streptococcus haemolyticus.

From this time cisternal punctures were done almost daily with drainage and occasional lumbar punctures always showed block. The first ten specimens of spinal fluid (to the fifteenth day of illness) gave positive results on culture and smear, but there was a slow rise in the sugar content, as shown in the chart.

In spite of all this the patient was neither toxic nor very ill. From the fifteenth to the twenty-fifth day the fluid was sterile, a total of eleven samples being obtained. The temperature fell after the fifteenth day and remained down except during two increases in the cell count of the sterile fluid. The last lumbar puncture showed a complete dynamic block, a clear yellow fluid, sterile, without cells, with a protein content of 1,200 mg. per hundred cubic centimeters and a normal sugar content.

The right knee jerk returned on the twentieth day, and neurologic examination gave negative results thereafter. The patient was up on the thirtieth day and was discharged as well on the thirty-eighth day. Hearing had returned, and the roentgenograms of the mastoid and chest were normal.

Two weeks after discharge from the hospital the patient reported to the outpatient clinic; she was well and had returned to work. In spite of the absence of symptoms a culture of material from the throat was still positive for Str. haemolyticus.

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* This and the following presentations were made by members of the departments of Neurology and Neurosurgery of the Massachusetts General Hospital.

The number of proved cases of pyogenic meningitis in which full recovery occurred is not large. There are about 15 reports in the literature in the last twenty years which concern a total of 94 cases, in 53 of which streptococci were cultured. B. coli was found in 2 cases, Staphylococcus in 7, B. influenzae in 20 and Pneumococcus in 12. The treatment used has been extremely varied, ranging from Bequeriel's puncture for orbital drainage to Zeller's insufflation of the meninges with acetylene gas.

Complete spinal block as one of the sequelae is rare. In 1930 Dr. John Hodgson, operating in a case of supposed tumor of the spinal cord at the eighth dorsal level in a man who had had meningococcic meningitis the year before, found dense adhesions with total block and a cystic cord. I wonder if the same syndrome may not develop in the case just reported.

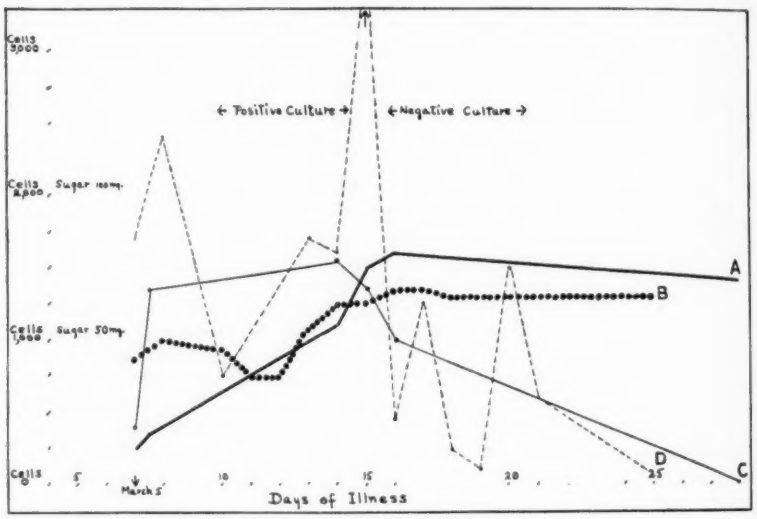

Chart showing the results of examination of the spinal fluid: *A*, the sugar content of the fluid obtained on lumbar puncture; *B*, the sugar content of the fluid obtained on cisternal puncture; *C*, the number of cells in the fluid obtained on lumbar puncture; *D*, the number of cells in the fluid obtained on cisternal puncture.

CHORDOTOMY FOR THE RELIEF OF PAIN IN TABETIC ABDOMINAL CRISES. DR. JOHN S. HODGSON.

I wish to report a favorable result of chordotomy performed on a patient with pain accompanying tabetic abdominal crises. The patient, an Italian waiter aged 43, married, has been a house patient at the Massachusetts General Hospital four times since July 1934. At the time of the first admission, July 23, 1934, he said that for the past ten years he had had periodic attacks of excruciating pain in the upper abdominal region on the left side. There was a history of chancre at the age of 18. The existence of syphilis was discovered ten years previously in a routine examination of the blood. Since, the patient has received extensive antisyphilitic treatment, including the administration of tryparsamide, intraspinal therapy, diathermy and malaria, with resulting negative Wassermann reactions of the blood and spinal fluid for several years.

The attacks of pain came every few weeks, with periods of freedom of only two or three weeks' duration. The pain was described as an intense feeling of fulness or as severe cramps. The attacks were accompanied by nausea, vomiting and "indigestion." Between attacks of vomiting there was much eructation of gas.

There were also constipation, urinary frequency, occasional blurring of vision and slight ataxia. Morphine had been used hypodermically twice daily for ten years, until two months previously, when he broke the habit at a state institution.

On admission there were Argyll Robertson pupils, absence of knee and ankle jerks, slight ataxia and some unsteadiness in the Romberg position. There were bands of hyperesthesia in two places on the left side of the body. On account of the pain the patient had not worked for three years.

On the first admission 1 cc. of a 10 per cent solution of phenol was injected into the fibers of the solar plexus paravertebrally, with immediate relief. The relief lasted, however, only four days. At this time a recurrent attack of epigastric pain was apparently precipitated by the use of a cathartic; there were accompanying nausea and vomiting. The attack disappeared somewhat more easily than those in the past, but moderate pain and irregular vomiting continued. Therefore, on August 18 alcohol was injected paravertebrally on the left side, with immediate disappearance of the pain, but again this was of a few days' duration. On October 18 the left splanchnic nerve and two sympathetic ganglia were resected retroperitoneally through a modified kidney incision. The old epigastric pain disappeared, but there developed a severe cramplike pain in the left lower part of the abdomen. Roentgenograms of the gastro-intestinal tract were taken at this time and were interpreted as showing gastritis and duodenitis.

The patient reentered the hospital on Jan. 18, 1935, with the history that since the last discharge he had continued to have cramping, agonizing pains in the left lower part of the abdomen, with the highest level at the umbilicus. There was no vomiting. On January 26 chordotomy was performed at the level of the first dorsal vertebra on the right side. This was followed by immediate and complete relief from pain. A level of analgesia to the nipple was obtained. No weakness of the limbs resulted; there was a temporary urinary retention, followed by slight incontinence, lasting a few weeks, but this eventually disappeared. When seen on various occasions, the last time three months after the operation, the patient was still entirely free from pain, had been working since immediately after discharge and was very cheerful.

The pain of tabetic crises has been variously ascribed to involvement of sympathetic or vagal nerve endings in the gastric wall, to involvement of the sympathetic plexuses, to irritation of a posterior root or ganglion or to the vagus centers in the floor of the fourth ventricle. The possibility of conduction of pain through afferent fibers in the anterior nerve roots has been mentioned. To this end various drugs, such as atropine and magnesium sulfate, have been tried, with but varying degrees of temporary relief.

Relief by surgical measures has been attempted in a number of ways for many years. This has varied from stretching the solar plexus to cutting the main trunks of the vagus nerves, cutting the splanchnic nerves or the posterior sensory roots, cutting the vagal endings in the wall of the stomach, combined section of the posterior and anterior nerve roots, sectioning the vagus nerve at the medulla, the von Gaza operation and, finally, anterolateral or spinothalamic section, known as chordotomy. Apparently no one method so far has been uniformly successful. On the whole, chordotomy probably offers the greatest chance of success and at the least risk. If, however, the crises in a given case are predominantly or partly vagal in type, chordotomy probably will not give complete relief. It is probable that each case should be carefully studied and analyzed, an attempt made to classify the condition as principally sympathetic or principally vagal and appropriate treatment based on this.

BRAINEDNESS AND HANDEDNESS: TWO CASES OF APHASIA. DR. EDWIN M. COLE.

It is often difficult to determine the handedness preference of a given family stock. Criteria of handedness are few and unsatisfactory, and histories from the lay observer are not always dependable, so that even when one wishes to take account of these factors one cannot determine what they are. Two unusual cases

of aphasia have been studied at the Massachusetts General Hospital recently, which seem to challenge some of the accepted theories in regard to the brainedness-handedness relationship.

CASE 1.—A retired cotton mill executive, aged 60, entered the hospital because of frequent attacks of weakness, faintness sometimes associated with a few convulsive movements and transitory loss of consciousness. The first such attack occurred twelve years ago, and during the past year attacks have become very frequent and more severe. For two years the patient's speech has been increasingly less free, and since December 1934 he has been able to say only a few words. He cannot read aloud, though he seems to enjoy looking through magazines and papers. His writing is a jumble of disconnected letters and words. Since October 1934 the right arm and leg have been clumsy. These limbs were weak and spastic as well as being hypesthetic. The tendon reflexes on the right side were more active than those on the left. A roentgenogram of the head showed an irregular net work of calcification in the left frontoparietal area, which measured about 4 cm. in diameter and lay opposite the middle meningeal artery. The pineal shadow was displaced downward and to the right.

This man was born left-handed and after some struggle was taught to write with the right hand, which he continued to use for this purpose. He did, however, perform many skilled acts with his left hand and maintained a fair degree of ambidexterity. As a young boy he stuttered, but this disappeared later. A careful review of the family history reveals no left-handedness in any relatives for two generations back. The patient's father stuttered in boyhood, and the patient's sister, though right-handed, claims that she can do more with her left hand than can the usual right-handed person. This same sister hesitates in speech a great deal, though she says she has never stuttered. Otherwise the family history is without significance.

CASE 2.—A man, aged 48, a mechanic and machinist, was well until two years ago, when he suddenly became unconscious for several hours. Thereafter he was unable to speak at all, nor could he read or write. There was hypesthesia over the right side of the body, as well as clumsiness and weakness. The reflexes on the right were more active than those on the left. There was right homonymous hemianopia. These signs and symptoms have all improved considerably, though the patient is still aphasic. This man was born left-handed, and his left hand has always been his hand of preference, though he has been able to use the right hand easily and in his work has used that hand a good deal. His mother and maternal grandfather were both left-handed. For two generations back no other history of left-handedness was obtained.

In these 2 cases of so-called left-handed persons, then, aphasia developed with lesions in the left hemisphere. Both had had a fair degree of ambidexterity. One had learned to write with the right hand, and the other had maintained his original hand preference. In 1 case the patient was the only left-handed member in the family in three generations, though there was a case of stuttering in the family. In the other case there was a definite strain of left-handedness in three generations.

What conclusions, if any, can be drawn from these 2 cases? Is there no relationship between handedness and brainedness? Does training in the use of the hands play a part in lateralizing the speech centers, or is heredity the important factor? These and similar questions come to mind in considering these 2 cases.

CONTINUED VASOCONSTRICTION AFTER SYMPATHETIC GANGLIONECTOMY CAUSED BY CIRCULATING EPINEPHRINE: A CAUSE OF THE POOR RESULTS OF SYMPATHECTOMY IN CASES OF RAYNAUD'S DISEASE OF THE ARM. DR. JAMES C. WHITE and DR. A. M. OKELBERRY.

Surgeons who have had a large experience with ganglionectomy in the treatment of Raynaud's disease have been forced to the realization that this operation is far less successful in the upper extremity than in the lower extremity. An explanation for the unsatisfactory results of cervicothoracic ganglionectomy was

first called to our attention in a patient whose hands within two weeks of a total sympathetic denervation became blue and cold on emotional stimuli. This woman had complete sudomotor and pilomotor paralysis, and therefore it is fair to assume that the vasomotor nerves were paralyzed. With the recent interest in chemical mediation of the nerve impulse, the possibility of residual vasoconstriction through the action of circulating hormones deserves consideration. Since the work of T. R. Elliott (1905) it has been known to physiologists that denervated smooth muscle becomes abnormally sensitive to circulating medulliadrenal hormone. This has been found to apply equally to the pupil (Meltzer and Auer, 1904), the denervated heart (Cannon, Lewis and Britton, 1926) and the central artery of the rabbit's ear (Meltzer and Meltzer, 1903). This phenomenon is excellently demonstrated in the work of Hartman, McCordock and Loder (1923). Smithwick, Freeman and White (1934) have shown that the same is true of denervated smooth muscle in the arterioles of man. Injections of physiologic amounts of epinephrine have caused a reduction in surface temperature in the fingers of 7 degrees F., with a fall of only 2 degrees in the toes.

Can this difference in reaction to the circulating hormone epinephrine explain the good results after ganglionectomy for Raynaud's disease of the legs and the poor results in the arms? Why should the vessels of the leg be less sensitive than those of the arm? Of course it is first necessary to prove that efferent sympathetic impulses have been interrupted as effectively by resection of the cervicothoracic ganglia and second thoracic ganglion as after removal of the lumbar ganglia from the second to the fourth. I must admit that an incomplete operation is often the cause of failure after denervation of the upper extremity, but we have demonstrated sudomotor and pilomotor paralysis and therefore presumably a successful vasoconstrictor paralysis in 6 cases of Raynaud's disease with residual vasospasm in the upper extremity. In addition, the denervated hand never remains as hot as the denervated foot. By inducing hypoglycemia with insulin we have demonstrated in these cases that a striking degree of vasospasm can be induced by secretion of the patient's own adrenal medulla.

Much can be learned about the physiologic responses of denervated arteries by experiments on animals. The rabbit is ideal for this type of work, because the central artery of the ear can be measured and photographed. Complete relaxation of vasoconstrictor tonus follows resection of the superior cervical sympathetic ganglion and the division of the sensory nerves at the base of the ear. This complete vasodilatation lasts only a few days. At the end of a week, which corresponds with the degeneration of the postganglionic vasoconstrictor neurons, the arteries in the denervated ear are again capable of full constriction. We have found that this occurs during activity and also in the presence of cold, pain and the subcutaneous injection of epinephrine in doses too small to affect the vessels of the normal ear. The denervated artery is dilated only when such an animal is at rest and relaxed, in addition to being warm. However, after resection of one adrenal and denervation of the other, the denervated arteries to a great extent lose their ability to constrict.

In striking contrast to the animals in which postganglionic sympathetic neurons have degenerated after resection of the superior cervical ganglion, interruption of the preganglionic fibers is followed by lasting freedom from vasoconstrictor tone. Section of the proximal motoneuron to the arteries of the rabbit's ear can be carried out either by resection of the stellate ganglion or by division of the upper second to the eighth pair of anterior thoracic spinal roots (Langley, 1892). The resultant degree of vasodilatation is striking and has been followed in our laboratory for months.

It is therefore evident that while a high degree of sensitivity of smooth muscle to epinephrine invariably follows degeneration of the postganglionic sympathetic neurons, this phenomenon is hardly noticeable after the preganglionic fibers have been destroyed (Hampel, 1935). The anatomic observations of Kuntz (1927) on the sympathetic rami to the brachial plexus and Adson's method of resecting the inferior cervical and upper two thoracic ganglia have made it possible to secure

complete sympathetic denervation of the arm. In spite of the anatomic effectiveness of this operation, resection of these ganglia must cause degeneration of the majority of the postganglionic vasoconstrictor neurons to the hand and therefore a high degree of sensitization to epinephrine. On the other hand, since the postganglionic neurons to the sciatic nerve originate in the sacral ganglia, resection of lumbar ganglia from the second to the fourth destroys mainly the preganglionic fibers to the foot. This I believe to be the secret of the divergent results in our present surgical treatment for Raynaud's disease of the upper and lower extremities.

INFECTIOUS POLYNEURITIS: PATHOLOGIC DEMONSTRATION. DR. CHARLES S. KUBIK.

The pathologic changes in 2 cases of infectious polyneuritis, similar to those described by Bashford and others, consisted of degenerative changes in myelin and axis-cylinders, proliferation of Schwann cells and infiltration with lymphocytes of the cranial and spinal nerve roots and peripheral nerves. Anterior and posterior roots were about equally affected, and there were marked cellular proliferation and infiltration in the spinal ganglia. Except for axonal reaction in the cells of the anterior horns and bulbar motor nuclei, there were no definite evidences of disease, such as lymphocytic infiltration or microglial proliferation, in the spinal cord or medulla. The blood vessels of the spinal gray matter were congested in 1 case but not in the other. The cerebral cortex and the cerebellum appeared to be normal. There was rather slight infiltration with lymphocytes and monocytes of the subarachnoid space.

Monkeys inoculated intracerebrally with emulsified spinal cord from each case did not show symptoms of disease. The animals were not killed.

THE NORMAL APPEARANCE OF THE SACRAL CANAL AS SHOWN BY IODIZED POPPY-SEED OIL. HENRY R. VIETS.

Following the injection of iodized poppy-seed oil 40 per cent, there is a great deal of variation in the appearance of the shadow outlining the lower end of the spinal canal under normal conditions. A number of tracings from roentgenograms showing the various types of shadow formation are shown in the accompanying illustrations: Although the shadow is large in *A*, this is due to the angle at which the picture was taken. There is no deformity in the actual outline. In *B* the shadow has taken the form of a long sharp cone, and a number of droplets have spread out on the sacral nerve. This is also a normal appearance. In *C* the iodized oil has sunk to the bottom of the spinal canal and shows a cone not quite as sharp as in the previous picture. Droplets have formed in the sheath of the fourth and fifth lumbar nerves, as well as along the sacral nerves. This appearance is characteristic of what occurs from twenty-four to forty-eight hours after the injection, or in some cases from two to three weeks later. The appearance in *D* is not dissimilar to that in the previous pictures but conforms more to a combination of *B* and *C*. Droplets of iodized oil have spread out more definitely along the sacral nerves. Although there was no clinical diagnosis other than lumbosacral strain, the appearance of the iodized oil in *E* is somewhat abnormal. One notes large masses of oil outside the usual cone formation, probably in the sheaths of the sacral nerves. These droplets, however, are larger than ordinarily occur and may indicate iodized oil in distended sheaths or possibly in the epidural space. Occasionally iodized oil supposedly put into the subarachnoid space by lumbar puncture is deposited in the epidural space when the needle is withdrawn or when it is not completely within the subarachnoid space at the time of the puncture. As in the preceding cases injections were all made into the cistern, it does not seem possible that the latter mechanism could be a factor in explaining the appearance here. The appearance is probably normal but cannot be stated as definitely so. In *F* the appearance of the iodized oil in the lower end of the spinal canal, with masses along the sheaths of the nerves, is normal. This shows another example of the varieties apparently within normal limits. The shadow in *G* is also normal

Tracings made from roentgenograms showing the types of shadow formation after the injection of iodized oil.

in appearance, but with the masses extending along the course of the sacral nerves, although seen in a roentgenogram taken at a higher level in the same case, showing the iodized oil striking down along the side of the spinal cord at the level of the eleventh and twelfth thoracic vertebrae. Some of it has passed out along the root lying between these two vertebrae. This is considered a normal appearance and is often seen in roentgenograms taken several months after the original injection. It is more common, however, for the iodized oil to pass out along the sacral roots than it is along those of the higher lumbar or thoracic. *I* presents an appearance similar to that in the other pictures, particularly *D*, but with more iodized oil in closer proximity to the sheaths of the sacral roots. There is no reason to consider that this is abnormal. The shadow in *I* was obtained in the case of a man, aged 37, with a ruptured disk between the fourth and the fifth lumbar vertebra on the left side. The indentation of the iodized oil shadow is clearly shown, and it has led to imperfect filling of the apex of the cone. The shadow in *K* was obtained in the case of a man, aged 30, with a ruptured disk in exactly the same area. A larger deformity of iodized oil shadow is shown, with more irregularity in its borders. *L* shows the shadow obtained in another case of ruptured disk between the fifth lumbar vertebra and the sacrum. A similar deformity of the iodized oil shadow is seen. Some of the iodized oil has sunk to the bottom of the canal and is passing out along the lower sacral roots. The last 3 cases were proved by operation and pathologic study.

TUMOR OF THE GASSERIAN GANGLION: REPORT OF A CASE. DR. WILLIAM JASON MIXTER and DR. CHARLES C. LUND.

Tumor of the gasserian ganglion is rare. Cooper in 1933 collected reports of 76 cases from the literature. This included all cases of tumor involving the ganglion that he could find. It seems probable that only a small proportion of these were growths arising from the nerve elements of the fifth nerve itself. No study has been made of the literature for this report.

We shall report a case of tumor of the fifth nerve involving not only the ganglion itself but also the second division as far as the skin of the face. On account of this it was possible to make a positive diagnosis prior to operation.

Mrs. I. S. L., was transferred to the Massachusetts General Hospital from the Huntington Memorial Hospital on April 20, 1934. The family history and the past history were unimportant. It is of interest, however, that the patient had had a nonmalignant mole removed from beneath the right eye. We believe that this was merely a coincidence. Ten months before admission to the hospital the patient noticed a mass the size of a pea beneath the outer corner of the right eye. Three months later she began to have lancinating pain about the eye. At this time the mass beneath the eye was excised in another city. The pain persisted, and she was given radium treatment, but without relief. About a month before she came under our care a small mass was noted in the scar, and subjective numbness was noted in the upper lip.

The patient was admitted to the Huntington Memorial Hospital and was operated on by one of us (C. C. L.). The mass was found to extend through the upper edge of the malar bone into the orbit beneath the eye. It was evident that the growth followed the course of a branch of the second division of the fifth nerve. It was not possible to excise it completely.

The pain in the face continued, and the patient was transferred to the Baker Memorial Hospital. Physical examination showed moderately elevated blood pressure and some evidence of myocarditis. Examination of the spinal fluid revealed an initial pressure of 60 mm. (water), no block, a total protein content of 38 mg. per hundred cubic centimeters and a colloidal gold curve of 0000000000. The Wassermann reaction was negative. Roentgen examination showed marked enlargement of the foramen rotundum on the right. Neurologic examination gave negative results except for an area of anesthesia on the right side of the face corresponding to the distribution of the second division of the fifth nerve.

At operation a purplish tumor of the gasserian ganglion was exposed. This mass appeared to invade the cavernous sinus; so the root sheath was opened and the posterior root was completely severed. No attempt was made to remove the growth.

Following this operation the patient had relief from the pain in the face and was in very good general condition for several months. Then she began to have slight, transient headaches. She reported at the hospital for follow-up examination on October 18, seven months after section of the nerve root, and died suddenly on the following day.

Autopsy revealed that death had occurred from rupture of the left side of the heart following an infarct. The tumor had increased slightly in size. Examination of the tumor and also of the specimen taken at operation by Dr. Charles Kubik revealed that the growth was different from the usual neurofibroma. His report follows: Microscopic sections of the trigeminal nerve reveal a tumor composed of cells with indefinite cytoplasm and long, rather large nuclei. The latter, lying in a fibrillar structure, varied considerably in size and shape, some being narrow and others long but quite thick, while many were pear-shaped or club-shaped. Several mitoses were observed.

A diagnosis of fibrosarcoma and of malignant neurogenic tumor (?) was made.

We believe that this case is of interest for several reasons: 1. Malignant tumors of the trigeminal nerve are unusual, particularly those apparently originating in the nerve or ganglion itself. 2. Enlargement of the foramen rotundum was demonstrated by a roentgenogram. 3. A positive diagnosis was made possible by the extension of the growth to the surface.

DISCUSSION

Dr. J. B. Ayer: Several years ago the society decided to have an occasional meeting at the Massachusetts General Hospital. These were not very successful because of poor attendance, but when Dr. McDonald suggested that the staff of the hospital give the papers of the evening it seemed wise to have a number of short papers on the work in progress. I hope that this method of presentation is satisfactory.

Dr. Schwab is to be commended for his persistence in treating patients with septic meningitis. Without using any new technic, he has demonstrated again the satisfactory results from repeated drainage of the spinal fluid. The organism must have been of low virulence, as the patient was ill for six days before treatment was begun; however, it is unusual under any conditions for a patient with streptococcic meningitis to recover. I do not see why he feels that a transverse lesion of the spinal cord is likely to develop, and yet it is possible that this may happen, as occurred in a patient with meningitis due to Staphylococcus albus whom I treated. I agree with him that a subsequent lumbar puncture should be performed to establish whether or not adhesions have formed.

With regard to Dr. Kubik's paper, I have the impression that the spinal cords showed very little damage. Is it not true that they usually show considerable? The picture referable to the pyramidal tract is there, and very early. I should like to know how much damage was found in these 2 cases. I was under the impression that more damage to the spinal cord than Dr. Kubik indicated was a rule.

A word or two about Dr. Viet's silhouettes: In spite of the fact that iodized poppy-seed oil has been used since 1920, the variations in the sacral canal shown by this substance are not well known. I think that the reasons are obvious: Until recently, the amount of iodized oil used seldom exceeded 1.5 or 2 cc., which is ample for localization of a tumor of the spinal cord. This amount, when seen in the sacral canal, shows the very bottom of the canal only. Now that amounts of 4 and 5 cc. are being used in localization of tumors of the cauda equina and ruptured disks, the sacral canal is beginning to be visualized a great deal more accurately. Incidentally, no ill effects are resulting from the larger amounts; indeed from the smaller amounts transitory irritative effects are observed.

DR. C. S. KUBIK: I did not happen to see the record of the case to which Dr. Ayer refers. The findings in these 2 cases seem to me to be essentially the same as those reported by others. There has been some difference of opinion as to whether changes found in the anterior horn cells represent simply an axonal reaction or the direct effect of disease. The absence of cellular infiltration or microglial proliferation in the spinal cord leads me to think that the cord is not directly affected.

DR. H. R. VIETS: There is little to be found in the current literature in regard to the outcome of cases of so-called "Jake palsy." Patients were first affected by this disease in 1930, and almost all of the incidents occurred during that year. It is now, therefore, five years after the initial infection, and one ought to be able to draw some definite conclusions at this time regarding prognosis. In all the cases that I have seen the paralysis of the hands has almost entirely disappeared, whereas the paralysis of the feet has only partially disappeared, and in some cases there has been no return of function. Why this should be is not at all clear. Perhaps Dr. White's remarks about the recovery rate of the hands as compared with the feet should be applied to this problem.

There is one other feature of "Jake paralysis" that has not been emphasized in the literature. In spite of the fact that the disease was supposed to be peripheral neuritis, spasticity developed in some of the patients as well. There is evidence, therefore, that the disease affected the pyramidal tracts as well as the peripheral nerves. Patients may, therefore, show feeble knee jerks and absence of ankle jerks but a bilateral Babinski sign.

A third point refers to recovery taking place after a long period of time. I have recently seen at the Long Island Hospital 2 or 3 patients who have stated definitely that recovery from foot-drop has taken place in the last few months, nearly five years after the onset of the disease. If this is so, and one can hardly doubt the evidence of these patients, must one not assume that regeneration of the more peripheral part of the nerves is taking place at the present time?

DR. B. T. BURLEY: Since Dr. Viets has mentioned my previous reports on cases of Jake paralysis I can confirm his statement as to the residual effects.

I have recently seen a striking case in which there was such contracture of the achilles tendons that the patient walked on his toes, with crutches. In my experience with these cases there was a marked difference in effect on the lower as distinguished from the upper extremities. Paralysis in the feet occurred two weeks earlier than paralysis in the hands and was always more severe. Recovery in the hands usually took place within six months. Many persons with severe involvement even now show only slight motion below the knee, and though there is a tendency to improvement, I fear that there will be no great relief from the paralysis of the legs.

DR. H. B. SANBORN: Cases of multiple neuritis of the type which Dr. Kubik has just reported may present an interesting and difficult problem clinically in differentiation from anterior poliomyelitis. A patient with such a condition was recently under my observation. Dr. Kubik did not enter so fully into the clinical course of his patient as I wish he might have done.

In my patient, a young man, the onset was what seemed an infection of the upper respiratory tract, followed a few days later by paralysis in the lower limbs. This soon involved the upper limbs and respiratory muscles. He was put in a respirator but died in spite of it, and no autopsy was obtained. A diagnosis of poliomyelitis of the ascending type could not be ruled out, but certain findings favor the condition's having been multiple neuritis of the type under discussion. The cell count of the spinal fluid, 28 lymphocytes, was consistent with either disease. The season of the year was not that at which poliomyelitis usually occurs. The white blood cell count of 15,000 was higher than is usually found. The temperature was from 101 to 103 F. for several days, and this also is unusual.

Book Reviews

The Brain as an Organ: Its Postmortem Study and Interpretation. By Frederic Wertham, M.D., and Florence Wertham. With introduction by Adolf Meyer, M.D. Price, $7.50. Pp. 538, with 166 illustrations. New York: The Macmillan Company, 1934.

This is an extraordinary book. The first 8 pages reveal the authors' message. The next 115 pages are a manual of laboratory technic, and the last 364 are a treatise on neurohistology. The emphasis of the latter section is first on the facts that "the brain reacts in very similar ways to the most varied kinds of damage. Its repertory of pathological responses consists of a relatively small number of units." Second, it is emphasized that the stage at which the lesion is examined is most important, and, finally, the distribution of the lesions must be accurately known. Most neurologists will readily accept and commend all this. The authors' main thesis is that "the brain as an object of histopathological study is also an organ of the body like any other, and not something unique, to be measured by entirely different standards." One finds it difficult to agree with this. In fact, the authors themselves refute it in the chapters on "Kinds of Lesions" and "Distribution of Lesions." They state: "It is not the multiplicity of damaging agents or of morbid processes which causes the great variety and complexity of different histopathological pictures. . . . . Most important is the stage in which we find the lesion." And further: "There was a period when 'fiber anatomical studies' devoted to problems of localization were so much in the foreground that the investigation of the nature of histopathological processes was greatly neglected. With the inauguration of finer histopathological studies a reaction set in against this tendency, and the details of the response of the tissue became the center of interest. This, in turn, was carried so far that one was satisfied with examining only a few samples of the tissue. In the time of Nissl, routine examinations were often limited to the study of one or a few small blocks from the cortex. No one would have thought of investigating so remote a structure as, for example, the substantia nigra, which in recent years has become such an important landmark in histopathology."

The reviewer holds that the most important cause of the complexity of lesions is the complexity of the nervous system; the brain is an organ *un*like any other, it *is* unique, and must be studied in a different way. True, there are but three basic types of cerebral tissue (glia, mesoderm and nerve cells), but the nerve cells are extraordinarily variable in structure, arrangement and metabolic activity; thus the substantia nigra, the nucleus ambiguus and the area striata cerebri (to take three of a hundred obvious examples) resemble each other so little that they are really different organs, histologically speaking. One sample of liver suffices as a histologic example of the whole organ; moreover, the liver is much alike throughout the phylogenetic mammalian series, whereas the brain differs greatly in different mammals and is unique in man. In other words, to consider "the brain as an organ" is a fundamentally false approach, because the brain is an intricate working combination of a hundred or more organs. This is the main reason why the brain "reacts with lesions which in our examinations seem to present such an enormous variety" (page 184).

Having controverted the authors' main premise, the reviewer contemplates the book more calmly and asks if it is worth while. On the whole, yes. Chapter I is a good lecture, important for all neurologists and psychiatrists. The authors state their reasons for writing the book on page 1, as follows: "Our own investigations have led us to a standpoint from which, we believe, it is possible to discern why the histopathology of the brain has reached what is being recognized more and more clearly as an *impasse*. Furthermore, they have suggested a simpler—and, at the same time, more realistic conception of the brain as an object of histopathological study. . . . Instead of expressing our point of view in the form of

theoretical affirmations and criticisms, we found it preferable—and indeed necessary —to present concretely the whole range of the fundamental data of the histopathology of the brain. The outcome is the present book. . . . The material discussed is derived from neuropsychiatric cases, from unselected cases of somatic diseases in general hospitals, and from animals with both experimental and spontaneous diseases. Some of it has not been published before. We have emphasized the significance of the non-neuropsychiatric material because of the too ready tendency of investigators to correlate with clinical symptoms any sort of microscopically demonstrable alterations they find in brains.

"To blend neuropathology more and more with a biologically oriented general pathology will be the task of the future—a task which we hope this book will further." This is the lesson preached. Certainly the "whole range of fundamental data" is not presented, and "affirmations" are not avoided. In fact, one finds it hard to agree with many of the dogmatic statements. But on the whole the lesson is good; perhaps it is too often repeated and too long drawn out; perhaps all the methodology might have been omitted and the neuropathology better arranged. Obviously the authors have made a courageous attempt at the impossible, but more power to them for it!

Neurology. By Roy R. Grinker, M.D. Price, $8.50. Pp. 979, with 401 illustrations. Springfield, Ill.: Charles C. Thomas, Publisher, 1934.

Grinker has made the laudable attempt to present neurology as a part of the biologic sciences rather than to recount symptoms and signs of disease. To accomplish this he has tried to give a survey of the embryology, anatomy, physiology, pathology and clinical semeiology of the central and peripheral nervous systems. Unfortunately, nine hundred and seventy-nine pages are scanty for this purpose, and some sections of the book have suffered as a result. On the other hand, this one volume may replace a textbook on neuro-anatomy and one on neurologic examination which the student might otherwise wish to acquire.

The chapter on "Technique of Neurological Examination" is good. There are the usual tables and figures showing the muscles that move the various joints, the levels of the important reflexes, the "motor points" and the sensory distribution of the nerve roots and peripheral nerves. The technic of the examination of patients in coma and of the examination of children is concisely and ably presented. The diagnostic value of lumbar puncture and examination of the cerebrospinal fluid is minimized, although the Fremont-Smith charts for differential diagnosis are presented.

The ninth chapter, on "The Spinal Nerves," deals with the anatomy of the peripheral nerves and the defects occasioned by lesions of them. Polyneuritis is discussed at some length, but little attention is given to the possible rôle of vitamin deficiency in the causation of the alcoholic and other forms of polyneuritis.

The sections on the cerebellum, posture and tonus and the cerebral cortex are among the best in the volume and present the student with the results of recent studies which are available nowhere else in such condensed form.

The chapters on tumors of the peripheral nerves and spinal cord (chapter X) and of the brain (chapter XVII) are excellent. These are profusely illustrated with diagrams, roentgenograms and photographs of patients. Bailey and Cushing's classification of the gliomas is followed, and many good photomicrographs are included. The chapter on the vegetative nervous system is a full and practical compilation of modern views on the subject.

Syphilis of the central nervous system is discussed in chapter XXII. There is a satisfactory classification. The subject perhaps deserves more extensive consideration, in view of the fact that syphilis is the most common disease of the nervous system. It is recommended that acute syphilitic meningitis be treated with iodides and mercury, although it has been repeatedly shown that the arsphenamines are more effective. The position is taken that vigorous early treatment of secondary syphilis may be the basis for the subsequent development of neurosyphilis. This doctrine appears unjustified in view of recent work.

Primary infections of the central nervous system are dealt with in chapter XXI. In view of the present state of knowledge, Grinker makes too sharp a differentiation between acute encephalomyelitis, postinfectious encephalomyelitis and multiple sclerosis.

The therapy of diseases of the nervous system might well be given more attention. For example, ephedrine is mentioned as a symptomatic remedy in the treatment of narcolepsy, but the dosage is not stated, nor is there any citation of excellent results obtained with this drug by various investigators.

The discussion of injuries of the head is more complete than in most textbooks of neurology. Not only the clinical manifestations, but also the pathologic changes, are well described. The principles of treatment are rather slighted, and, indeed, this is a criticism which could be made of the book as a whole. Considering the importance of surgery in modern neurology, the collaboration of a trained neurosurgeon would add to the value of the text. For example, section of the eighth nerve is described as a peculiarly difficult operation, when as a matter of fact it is rather easily accomplished by skilled hands.

The form of the book is good. The illustrations are clear and serve a good purpose. A list of representative references is given at the end of each chapter, and there is a fairly complete index. No attempt is made to invade the field of psychiatry. The style is forceful, even somewhat didactic. Its clearness is marred by a rhetorical infelicity in many passages. Emphasis is given to the author's own experiences, which is, on the whole, an advantage. It is a book which may be recommended to medical students, and is also valuable as a reference work, as it assembles from widely scattered sources a large body of data not found in other textbooks. If it is less complete in its account of clinical syndromes than other textbooks, it provides an unusual background of fundamental information and stimulates original thought.

Handbuch der Neurologie. Edited by O. Bumke and O. Foerster. Volume 7, part 1. **Humoral Pathology of Nervous Diseases.** Berlin; Julius Springer, 1935. Price, 87 marks. Pp. 505, with 67 illustrations.

This volume is by Prof. F. Georgi and Prof. O. Fischer. The first part is concerned with a general consideration of the blood, urine and spinal fluid. The latter part deals with changes in the spinal fluid in various nervous diseases.

The general section dealing with the blood and urine covers thoroughly the work on these body fluids from the chemical, physical, colloid and immunologic standpoints. It is an exhaustive review which deals with the chemistry of these substances very completely. The spinal fluid is also covered exhaustively. There is an excellent general discussion of the cells, specific gravity, viscosity and other physical characteristics, the inorganic and organic chemical constituents and the biochemistry and immunology. Some idea of the completeness of the review is gained from the fact that 300 pages are devoted to a general discussion of the blood, urine and spinal fluid, 100 of which are taken up with a consideration of the fluid alone. About 75 pages are devoted to abnormalities of the spinal fluid in various diseases.

While little that is specific is known of the blood and urine in nervous diseases, a review such as is presented in this volume is not only useful but valuable. The section on the spinal fluid is excellent. This volume is a fine contribution. In itself it is of value for all who wish to keep abreast of the more dynamic aspects of neurologic disorders.

Handbuch der Neurologie. Edited by O. Bumke and O. Foerster. **Diseases of the Spinal Cord and Brain.** Berlin: Julius Springer, 1935. Price, 98 marks. Pp. 575, with 24 illustrations.

The first article in this volume is by Kinnier Wilson on the epilepsies. It is well considered and reflects the wide clinical experience of its distinguished author. The volume is worth owning for this article itself. It is in English.

Narcolepsy is covered by Wilder with the help of material gathered by Redlich. The subject is completely treated.

One of the best articles in the book is that on vosomotor trophic diseases by Cassirer and Hirschfeld. This is essentially a revision of the article by Cassirer written for the Lewandowsky *Handbuch* in 1914. It is extremely well done. There is a good analysis of Raynaud's disease. There is also a good section on Quincke's edema. Other vasomotor disturbances, such as acrocyanosis, erythromelalgia, acroparesthesias, sclerodermo and facial hemiatrophy, are covered well.

Spasmophilia is covered by Husler. Richter contributes a section on migraine. There is a section on the neurasthenic reaction by Braun and one on the so-called organ neuroses by Jahrreiss.

The volume is a good one and can be highly recommended.

Die Mangel-Diagnose als Voraussetzung für die Ermöglichung seelischer Gesundung. By Franz Schauer. **Heft 28. Arzt und Seelsorger.** Price, 1.20 marks. Pp. 32. Schwerin: Friedrich Bahn, 1934.

Schauer claims to be expounding "a diagnostic and therapeutic fundamental principle" that is at the basis of mental ailments and must be the basis of their cure. He states that he refers to a group which in the psychotherapeutic literature up to now has been designated as nervous, neurotic, psychoneurotic, criminal or made up of persons with character difficulties. They all suffer, he believes, from "active pessimism"; that is to say, they think only of their symptoms and difficulties. The real trouble is that they lack a natural "positive" point of view. Schauer admits that this is a simple theory. "Mental 'diseases' are deficiency diseases." The patient fights against the "negative" instead of being interested in the "positive." This "correct diagnosis" alone permits the psychotherapist to effect a cure. Unfortunately, the author does not tell how it is done.

CORRECTION

The news item on Page 1130 of the November 1935 issue of the ARCHIVES OF NEUROLOGY AND PSYCHIATRY contains an error. The study that was contemplated was of homosexuality as it exists among the members of the United States Merchant Marine rather than of the United States Marine Corps.